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SPINAL LOCALISATIONS AS INDICATED BY SPINAL INJURIES.

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In 'BRAIN' for January, 1887, I published a series of nine cases of injury to the cervical region of the spinal cord, observed during the previous eighteen months in the Manchester Royal Infirmary, and was able to draw therefrom certain conclusions, which would appear to be of interest from the points of view both of physiology and of pathology. Since then I have had a further series of ten cases, which enable me to confirm and to extend the results then arrived at, and which I now bring forward as illustrating more especially the arrangement of the various nuclei situated in the brachial enlargement of the spinal cord. I have again to thank the Infirmary staff for permission to use these cases. They consist of seven fatal instances of fracture-dislocation, one of dislocation, which was cured by early reduction, and two of hæmorrhage into the spinal cord. My primary object in watching and recording them having been to ascertain more accurately than has hitherto been done the distribution of the paralysis resulting from injuries at different levels of the cord, I have arranged the first seven (fatal) cases in anatomical order, followed by the three others in which the seat of the lesion was inferential only, and have especially dwelt upon the exact distribution of the paralysis, but I have also referred to numerous other points of interest which they present. Further, this paper must be considered

as a continuation of the one above spoken of, to which frequent reference will be made.

Going back then at the outset to the above-mentioned paper, I would recapitulate some of the results then arrived at. I presented the following cases:—1 (pp. 515 and 516). Two cases of fracture-dislocation between the fourth and fifth cervical vertebræ, in which all the muscles of the upper limbs, with the doubtful exception of the supra- and infra-spinati, were paralysed. 2. (p. 511). A case of fracture-dislocation between the fifth and sixth cervical vertebræ, in which all the muscles of the upper limbs were paralysed *except the deltoid, biceps, brachialis anticus, and supinator longus*

FIG. 1.—Position occupied by the limbs in a case of complete transverse destruction of the spinal cord immediately below the level of origin of the fifth cervical nerves.

and again doubtfully the supra- and infra-spinati, and in which the patient's arms consequently persistently maintained the position indicated by the photograph of which an engraving is here reproduced. And 3. Two cases (pp. 518 and 520) of fracture-dislocation between the fourth and fifth cervical and fifth and sixth cervical vertebræ respectively, in which, owing to the obliquity of the crush of the spinal cord, the fifth cervical nerve-root escaped on the right side only in the one case and on the left side only in the other, the result being that in the former the deltoid, biceps, brachialis anticus, and supinator longus escaped being paralysed on the right side, and in the latter they escaped on the left side only; the limb thus partially spared presenting in both instances a position

similar to that of Fig. 1, whereas the opposite limb, whose fifth cervical root was involved in the lesion, was entirely flaccid.

Hence it was proved that, in injuries involving the cord below the fifth cervical nerve-root, we find the upper limb presenting a condition which is the exact antithesis of "Erb's paralysis" of the adult, or Duchenne's "obstetrical paralysis" as seen in the infant, and that consequently the limb occupies in such cases a position of abduction at the shoulder, with flexion of the elbow and supination of the wrist. It was further concluded, for reasons to which it is unnecessary again to refer, that the fourth cervical nerve innervates the supra- and infra-spinati muscles. These conclusions the present paper will be found to confirm, but we shall now also be able to trace the arrangement in the brachial enlargement of a number of the other spinal centres for the various muscles of the upper limb. In endeavouring to work out this matter I have entirely ignored the results as to localisation previously arrived at by other methods, and have confined myself entirely to the evidence yielded by my own cases, the more so as the conclusions are consistent with the majority of previous observations.

Before proceeding to give the evidence, I shall now tabulate provisionally what I am so far led to believe to be the arrangement, from above downwards in the cord, of the muscle nuclei, as follows:—

Supra-spinatus and infra-spinatus Teres minor (?)	4th cervical nerve.
Biceps Brachialis anticus [1] Deltoid Supinator longus Supinator brevis (?)	5th cervical nerve.
Subscapularis Pronators Teres major Latissimus dorsi Pectoralis major Triceps Serratus magnus	6th cervical nerve.
Extensors of the wrist	7th cervical nerve.
Flexors of the wrist	8th cervical nerve.
Interossei Other intrinsic muscles of the hand	1st dorsal nerve.

[1] I have not identified the brachialis anticus, but from Erb's researches and others, assume it to be in close connection with the biceps.

The various cases which follow will, I believe, substantiate the above arrangement.

Case 1.—A man aged 35 was admitted under Mr. Heath's care on June 18, 1887. He had the night before admission fallen down about ten steps on to his head, as evidenced by bruises on the left side of the cranium. As the man died in a few hours, I was obliged to obtain my information from Mr. Thompson, the house surgeon.

At the back of the neck was a large amount of effused blood, but the spines of the vertebræ were distinctly felt, that of the sixth cervical being apparently displaced slightly to the right. The head itself was not twisted, and could be turned from side to side, nor did the neck appear abnormally flexed or extended. There was only slight pain in the back of the neck.

The lower limbs and trunk were completely paralysed; the arms were held rigid with the elbows abducted, and the hands lying on the epigastrium extended and prone. Anæsthesia was complete below a point about two inches above the nipples, being also complete over the shoulders and in the upper limbs. The pupils were reduced to fine points, but the size of the palpebral fissures was not noted. Reflexes were not observed. The urine was retained, and the penis turgid.

As the man was being placed in bed respiration became slow and gasping, with distinct action of the scaleni and sterno-mastoids; the patient grew cyanotic and respiration then stopped, the pulse ceasing about half a minute later. There was no *post-mortem* examination.

The above case does not carry us very far, but it is clear from the facts adduced, and especially from the anæsthesia affecting the whole surface of the upper limbs, that almost the entire brachial region was affected. With the exception of the abduction of the shoulders and flexion of the elbows, the arms were in the position of rest, resulting from complete paralysis. Hence then the biceps and deltoid had escaped, but the supinators and all muscles mentioned below them in our table were paralysed; that is to say, the hæmorrhage resulting from the crush of the cord had extended partially into the territory of the fifth root, destroying the centre, at its lower part, for the supinator, but sparing the biceps and deltoid above.

Case 2.—A. B., aged 23, was admitted to the Manchester Infirmary under the care of Mr. Jones on April 7, 1888. He was playing football, and, while stooping forwards with his head thrown up in order to "tackle" an opponent, came into violent collision, his head being jerked backwards. He was immediately paralysed.

On admission the spine of the seventh cervical vertebra was felt to be distinctly depressed, but with little or no lateral displacement. There was a good deal of swelling about the back of the neck, and the patient complained of great pain. The lower limbs and trunk were completely paralysed, respiration being diaphragmatic. In the upper limbs the only muscles not paralysed were the flexors of the elbow. They at first lay extended and close to the body, but the patient bent the elbows so as to bring the hand up to the shoulder when told to try to move them. The pupils were slightly contracted.

An attempt was at once made to reduce the dislocation by extension, followed by flexion of the neck, and a distinct jerking was felt by the assistants who were steadying the neck. The patient expressed himself as feeling his pain relieved, but no other change was produced. Later on another attempt at reduction was made with chloroform, but with equally little success. The patient was now put to bed, the head being steadied by pillows and sandbags. At this time the temperature was below 95° and the skin felt cold; the pulse was strong and regular; the penis was turgid, the urine retained.

About 4 A.M. on the following day, the temperature began to rise rapidly; the skin was hot, flushed, and perspiring; and the patient became delirious. At 6 A.M. the temperature was 106°·6, at 8 A.M. 108°, and at 9.30 110°, at which time he died comatose.

There was no *post-mortem* examination. For notes of the above I am indebted to Mr. Stocks, the house surgeon.

This case is also somewhat unsatisfactory, as the accident happened on Saturday night, and the man died on the Sunday morning, so that it was not very fully observed. It indicates sufficiently the high position of the nucleus for the flexors of the elbow, and shows that the deltoid and supinators, also supplied by the fifth root, were paralysed. Thus, then, we are led to place the deltoid nucleus below that of the flexors of the elbow, a conclusion confirmed by a case mentioned in my previous paper, where at first the entire fifth root was spared, but where, as myelitis extended upwards, "the contraction of the deltoid muscles, which had maintained the abducted

position of the arms, ceased, and the elbows fell to the sides, the elbow-joints themselves remaining flexed."[1] These two cases, then, compared with the cases referred to in the previous paper, carry us so far as to assign the order—

{Biceps.
{Brachialis anticus.
Deltoid.
Supinator longus.

Case 3.—J. C., a labourer, aged 38, was admitted to the Manchester Royal Infirmary under the care of Mr. Hardie on October 25, 1887. On the afternoon of this day he had fallen from a waggon for a distance of about six feet, striking the back of his shoulders and head. He was immediately paralysed, but did not lose consciousness.

I did not see him until the following day, but am informed by the house surgeon that his condition on admission was the same as when he came under observation.

On Oct. 26 he had pain across the shoulders, shooting down the arms to the elbows, but no tenderness of the spine or pain on pressing down the head. There was some arching backwards of the cervical vertebræ, but no lateral deviation and no obvious deformity of the spinous processes.

The lower limbs were absolutely paralysed, as were the abdomen and thorax, respiration being diaphragmatic, jerky, 18 per minute, with subjective sense of dyspnœa and cough. The left upper limb was abducted at the shoulder, with the elbow flexed and the hand across the chest, the right being completely flaccid. On the left side he had the power of voluntary contraction of the biceps, supinator longus, and deltoid, but not of the other limb muscles, whereas on the right side all the muscles were paralysed. On both sides the pectorals and latissimi were paralysed, but on the left side the supra-spinatus and infra-spinatus could be made to contract, whereas on the right they could not. Hence of the intrinsic and extrinsic muscles of the upper limb all were paralysed on the right side, but the spinati, deltoid, biceps, and supinator longus had escaped on the left, giving the limb the above-described characteristic position. The neck muscles were normal on both sides. Both palpebral fissures and pupils were smaller than usual, but not so markedly as in many cases.

Anæsthesia extended as high as the third rib in front and the

[1] As the above is passing through the press, I have seen another case in which ascending traumatic myelitis caused paralysis of the deltoid *before* that of the biceps, a condition, I admit, at first considered improbable.

sixth cervical spine behind, but on the right side he retained sensation over the region of the deltoid and slightly beyond it, and on the left side it extended over the area of the deltoid and thence down the outer side of the limb to about the root of the thumb, becoming, however, very vague at the lower part. The anæsthetic boundary was not very sharply marked, and he had no hyperæsthesia.

The knee-jerk and plantar, cremasteric, gluteal, and epigastric reflexes, were absent. The skin felt dry and warm, although the temperature was normal. Urine was retained. The penis was turgid, but less so than on the previous day. Pulse 66, feeble, and with a very marked respiratory wave, being full and soft towards the end of inspiration and very small towards the end of expiration.

The man's condition being clearly otherwise hopeless, Mr. Hardie determined, after consultation with several of his colleagues, to "trephine" the spine, which was done at 4.20 P.M. on the afternoon of this day, about twenty-four hours after the injury. For the subsequent notes, I am indebted to Mr. Bannister. Chloroform having been administered, a vertical incision about four inches long was made over the cervical spines, having its centre opposite to the fifth. The muscles being cleared from the laminæ, an interval of about a quarter of an inch was found between the fifth and sixth spines, and the fifth vertebra appeared to be slightly displaced forwards. The laminæ of the fifth and sixth were now removed by bone forceps, when the dura mater was exposed, presenting a perfectly normal appearance without any trace of hæmorrhage. As nothing further could be done, the dura was not opened, but the muscles were brought together by deep catgut sutures, a large drainage tube inserted, and the wound closed over a smaller superficial drain. The carbolic spray was used throughout, and wood wool pads used as a dressing. After the operation brandy was given at frequent intervals.

At 6 P.M. the pulse was 54 and feeble; temperature 96°; respirations 13, and still purely abdominal. At 10 P.M. pulse 74; temperature 98°·6, respirations 16. The patient passed a fairly good night, but in the morning the temperature was 103°·4. About 3.20 P.M. on the afternoon of the 27th he became suddenly much worse; the respirations became very feeble and then stopped, the abdomen being uniformly distended; the heart-beats, which were very feeble and infrequent, continued for about ten minutes after respiration had ceased, but stopped at 3.35. At 3.40 the temperature in the mouth was 104°, and the body, which had previously felt very hot, was already noticed to be beginning to cool.

The *post-mortem* examination was made by Dr. Harris. The disc between the fifth and sixth cervical vertebræ was found to be ruptured, the former bone projecting very slightly forwards. No fracture was discovered. The dura mater was uninjured, but the cord was flattened opposite the seat of injury, and was much contused for about an inch above and below, containing hæmorrhages in its substance and in the central canal; elsewhere its structure was normal. The first and second bones of the sternum were also partially separated, and the lungs much congested.

This is another case similar to those already mentioned, in which the cord was crushed just above the sixth cervical root, but on the right side the fifth root was also affected. Unfortunately, I was unable to be present at the *post-mortem*, and the exact pathological cause of this obliquity was not noted, but doubtless the hæmorrhage which occurred in the substance of the cord extended somewhat higher on the right side. The case merely confirms our previous conclusions.

Case 4.—J. E. C., aged 33, was admitted into Mr. Heath's wards on March 25, 1887, at 2.30 P.M. He had shortly before been "larking" with some friends, and had another man seated upon his shoulders, when he was pushed backwards in the face against a high counter. The result was that his neck was twisted backwards, the man falling from his shoulders. For the first few minutes he only noticed pain in the back of the neck, but then his legs began to feel weak and he lay down: within ten minutes the lower limbs were completely paralysed and insensitive, and he found that he could not straighten the left fore-arm.

When admitted, there was paralysis of the lower limbs, and the left upper limb was only partially movable, being flexed at the elbow and somewhat abducted at the shoulder. The right limb appeared to retain more power. The temperature at 4 P.M. was 94°·2, at 8 P.M. 97°·6, at midnight 99°·2, near which point it remained for some days. He was placed on a water bed and the urine withdrawn by the catheter.

On the following morning he complained of pain in the back of the neck, but there was no local deformity, and no pain on pressing down the head. The lower limbs were absolutely paralysed, as were the abdominal and thoracic muscles, respiration being diaphragmatic.

The left upper limb lay with the fingers flexed, the wrist

straight, hand prone, elbow flexed, and the humerus abducted to an angle of about 45° with the body: he often raised the humerus above the shoulder, otherwise retaining the same positions, but with the wrist becoming semi-prone. He could not extend the elbow nor flex nor extend the fingers, but on attempting to flex the fingers there was slight extension of the wrist; he had also slight power of flexion and extension of the wrist, but this movement was extremely limited: pronation and supination were fairly good: the pectoralis major, latissimus dorsi, and apparently the subscapularis contracted very feebly; the deltoid, biceps, and supinator longus were but little if at all impaired.

On the right side the condition was practically the same, except that there was rather more power in the wrist. On taking a deep breath, the sterno-mastoids, trapezii, and levatores angulorum scapularum contracted very distinctly.

As regards sensation, there was "a feeling of heat" in the left palm and fore-arm. Anæsthesia was nowhere absolute, but was almost so below the third ribs, and in the upper limbs internally to a line running down the centre of the arms and fore-arms to the styloid processes of the ulna, both before and behind. Below these limits there was absolute analgesia, but a vague sensation was conveyed by tickling. The limit was very ill-defined, and above it was an indistinct hyperæsthetic zone.

There were no superficial reflexes nor tendon reactions.

Urine was retained, and had to be drawn off by the catheter: its sp. gr. was 1024; it was neutral, and contained neither sugar, albumin, nor any deposit. The bowels had not been moved since admission. The respirations were at the rate of 19 per min., with no sense of dyspnœa, but slight cyanosis. Pulse 69, full and soft. Skin dry and warm. The penis was turgid. Both palpebral fissures and pupils were small, and there was no dilatation of the pupils on pinching the neck.

Two days later, the paralysis of the upper limbs was of equal extent on both sides. There was complete loss of power in all the intrinsic and extrinsic muscles except the deltoid, biceps, supinator longus, and subscapularis, the elbows being flexed, the humeri abducted but not rotated outwards, and the hands thus lying across the chest. The pectoralis major, latissimus dorsi, and teres major had ceased to act at all. The region of anæsthesia in the upper limbs had also extended. The temperature was normal. The optic discs were examined, but presented no abnormality. Priapism was less marked.

On the 4th day the urine contained a little pus (and therefore albumin), but no sugar. Its reaction was acid.

On the 5th day the optic discs appeared less well defined than before, and presented some venous congestion.

On the 6th day the subscapularis and supinator longus could no longer be felt to contract, but the arms usually occupied the same position as before, although when in sleep they were often raised above the shoulder. He complained much of a sense of pain and stiffness in the upper limbs. The optic discs were more vascular and were hazy—an observation which was confirmed by Dr. Little, who held, however, that there was no neuritis.

For a few days sensation in the trunk and lower limbs now seemed to improve slightly, but the patient became weaker, and was frequently somewhat delirious at night. The temperature ranged from 100° to 101°.

On the 11th day were noted tremors of the paralysed muscles of the upper limbs. A day later, the biceps and deltoid of the left side were found to be beginning to fail, and the humerus was less abducted than formerly. At the same time sensation began to fail again.

On the 14th day the left upper limb was absolutely paralysed and anæsthetic, the humerus lying by the side, the elbow at a right angle. On the right side, the only muscle whose contraction could be felt was the deltoid. Ecchymoses had formed on the heels, toes, and malleoli.

On the 15th day we found distinct knee-jerk on the right side, and on the left a slight reaction could be obtained.

A few days later he developed symptoms of pneumonia, and the condition of the urine became much worse. The temperature, which had regained the normal on the 15th day, again began to rise on the 23rd, keeping a fairly steady upward curve until April 23rd or the 29th day, when it reached 104°·6 and when he died. During the latter period there appeared to be more wasting in the fore-arms and hands than in other parts of the body. On several occasions also it was noted that the passage of the catheter was accompanied by reflex contractions of the sartorii.

At the *post-mortem* examination I found some separation of the fifth and sixth cervical spinous processes. The body of the fifth was partially dislocated forward, forming a very obtuse angle with that of the sixth, and overlapping the latter by about a line anteriorly. The articular processes were *in situ*. The upper and anterior margin of the body of the sixth vertebra was very slightly ground off, but there was no other fracture. The vertebral canal contained a little dark clotted blood at the seat of the lesion. The cord was here partially compressed and very pale, all distinction

of white and grey matter being lost. This condition extended only for about an inch, and elsewhere the cord presented no macroscopic abnormality. No nerve-roots were crushed. There were marked cystitis and double pneumonia.

We have here another case in which the cord was crushed below the fifth cervical nerve-roots, the muscles supplied by which retained their power for a considerable time. The case differs, however, from those previously mentioned in that the annihilation of the functions of the cord below the lesion was less complete, as evidenced by the fact that anæsthesia was not absolute, and that some power remained in muscles supplied by nerve-roots distinctly below the lesion. The examination on the second day showed that, whereas the deltoid, flexors of the elbow, and supinators retained a very fair amount of power, the other muscles of the limb were paralysed to a varying extent. Thus next in order of power we find the subscapularis, pectoralis major, latissimus dorsi, and pronators, between which no distinction could be drawn, and we find also that the extensors of the wrist still retained a little power. On the fourth day we have paralysis of all the muscles of the plexus, except the "fifth-root group" and the subscapularis, the latter preventing the outward rotation of the humerus seen in Fig. 1. Again, on the sixth day the subscapularis and supinator fail, and later the deltoid and biceps begin to do so. As there can be little doubt that myelitis was extending upwards, we may then venture to give the following as the order in which the muscles here lost their power:—

1. Intrinsic muscles of the hand and flexors of the wrist immediately completely paralysed.

2. Extensors of wrist almost absolutely paralysed on second day.

3. Pectoralis major, latissimus dorsi, teres major and pronators following before fourth day.

4. Subscapularis retaining power longer than any muscles but those of the fifth root.

This, it will be found, is in accordance with our scheme of localisation. The position of the pronators is the most doubtful and the most difficult to ascertain, as the action of gravity interferes with the attitude of the hand.

Case 5.—E. M., a woman aged 43, was admitted under the care of Mr. Heath on December 5, 1886, on which day about 11 A.M., while intoxicated, she had fallen down some twelve steps. She was unconscious for a short time, and on recovering found that she had lost all power of moving the trunk and limbs. She was brought to the Infirmary, and admitted about 5.15 P.M. on the day of the accident.

On admission she was very drunk, and a complete examination was impossible. The skin felt very cold and the temperature was below 95°, but no thermometer of a lower register was at hand. The pulse could not be felt. There was no sweating. She complained of pain behind the shoulders, but there was no deformity. The lower extremities and the left upper limb were entirely paralysed and flaccid. On the right side the shoulder was abducted, and rotated somewhat outwards, the elbow being flexed, the wrist and fingers flaccid. Respiration was diaphragmatic. The urine was retained. Owing to the vagueness of her answers, it was impossible to ascertain the limits of sensation, but there appeared to be anæsthesia below the neck. The right pupil was small, distorted, and immovable, owing to posterior synechia; the left was small, but responded both to light and accommodation.

When I examined her on the following morning, the patient presented the following symptoms:—The intellect was perfectly clear, and she gave rationally the above account of her accident. She presented total paralysis of all four limbs, and of the muscles of the chest and abdomen. She had no power of moving even the shoulders; the pectoralis major, deltoid, supra- and infra-spinati, latissimus dorsi and subscapularis, as well as the intrinsic muscles of the upper limb, being quite flaccid on both sides. There was no tendency to rotation of the humerus, either inwards or outwards. On the other hand, the trapezii and sterno-mastoids were tonically contracted.

Respiration was diaphragmatic, but there was no indication of dyspnœa and the voice was not weak. The urine had to be drawn off by means of the catheter, and the bowels had not been moved since admission. The palpebral fissures were smaller than usual, giving the patient a sleepy look. The right pupil was as described above, but presented some further contraction on exposure to light.

She complained of a sense of coldness in the left foot, and of great pain in the back of the neck and across the shoulders, pain being aggravated by any movement of the shoulder-joint. The limbs and trunk were completely anæsthetic, there being a band of hyperæsthesia about $1\frac{1}{2}$ in. wide, extending in the form of a

collar across the chest, at the level of the second rib and outwards across the deltoids. Above this line sensation was normal. It was not possible accurately to define the limit of sensibility behind, owing to the necessary movement causing great pain in the neck. The firmly-contracted trapezius was painful on deep pressure. No pain was caused by very firm downward pressure on the head.

The plantar reflex was exaggerated, the slightest tickling of the sole causing clonic spasms of the limb. No abdominal cutaneous reflex could be obtained. There was no knee-jerk, nor ankle clonus.

The cranial nerves presented no abnormality. The right optic disc was normal; the left could not be seen, owing to a commencing cataract.

The pulse was very irregular and intermittent, full, soft, and beating at the rate of 108 per minute. The heart-sounds presented nothing abnormal.

Respirations were 28, and, as above stated, entirely diaphragmatic, the chest falling in on inspiration.

The temperature at 8 A.M. was 95°. When examined at 12 midday, it was 97°, but the skin of the trunk and lower limbs felt warm and somewhat dry.

The urine had a sp. gr. of 1024, was acid, contained no albumin and no deposit. It decolorised but did not precipitate Fehling's solution. The nurse informed me that she had a considerable amount of vaginal discharge.

The temperature now began to rise steadily, and at 9 P.M. on the same day reached 101°·6, at which it remained until death, which occurred at about 3 A.M. on the following day; that is, about forty hours after the accident. Death resulted from failure of respiration.

At the *post-mortem* examination we found a separation between the laminæ of the sixth and seventh cervical vertebræ, the whole of the ligaments uniting the arches being ruptured, and the sixth vertebra being carried forwards and to the right.

On removing these bones, the left inferior articular process of the sixth was found completely slipped off the superior articular process of the seventh, whereas on the right side it was only displaced forwards to a slight extent. There was an obliquely horizontal fracture across the body of the seventh vertebra, extending from the cartilage above it forwards and slightly downwards to a line about one-fourth of the distance from its upper to its lower border in front. The piece of bone thus separated was, with its adjacent cartilage and the sixth vertebra, carried forwards and somewhat downwards and to the left. On opening the spinal canal there was found extravasated and coagulated

blood external to the dura mater, throughout the entire distance from the sixth cervical vertebra to the inferior termination of the canal. The disc between the sixth and seventh vertebræ had been crushed and forced backwards into the spinal canal, forming a shelf about one line in depth, which pressed upon the cord, but not to any great extent. The roots of the seventh nerves were crushed. The cord was slightly pressed upon by the above-mentioned projecting disc, but was nowhere compressed by the extra-meningeal hæmorrhage. Opposite the body of the sixth vertebra it presented a slight swelling of a bluish colour. Its substance was here quite diffluent and full of dark blood. From this point upwards as high as the origin of the third cervical nerves the cord was very soft and contained small extravasations of blood, mainly in the grey matter and especially in the central canal, these appearances being less marked at the upper than at the lower part. Below the sixth intervertebral disc the cord was slightly softened for the distance of about an inch. Elsewhere it presented no abnormality to the naked eye. The membranes appeared normal.

In this case no information of value is to be obtained as to the question of localisation, as the hæmorrhage into the cord extended so rapidly as almost at once to paralyse the cord throughout the entire brachial region, although the lesion of the vertebræ was at the level of origin of the seventh cervical nerves. For a short time after admission the fifth cervical nerve on the right side escaped, but this also was destroyed before I saw this patient, and death soon followed. The extensive central hæmorrhage found at the *post-mortem* amply explains this condition, and the case is chiefly of use as showing that the damage to the cord may almost at once extend to a considerable distance above the vertebral lesion.

Case 6.—W. T., aged 28, a labourer, was admitted to the Manchester Royal Infirmary under the care of Mr. Wright on October 3, 1887. Two days previously he had been at work on the roof of a house when he fell, a distance of about eleven feet, on to a block of stone, which struck him just between the shoulder blades, causing his head to be jerked backwards. He felt very sick, but did not vomit, and then found that he was powerless to move either his arms or his egs. During the two days which he remained at home no change can be ascertained to have occurred in his condition.

On admission he presented complete paralysis of the lower limbs and of the abdominal and chest muscles, respiration being diaphragmatic. There was pain over the first dorsal vertebra, but not elsewhere, and no pain on pressing down the head. The upper limbs lay across the chest with the elbows close to the sides, and flexed, as in the annexed sketch made by the house surgeon, Mr. Bannister. On both sides the flexors of the wrist and fingers were completely paralysed, and he had no power of grasping; on the right side the extensors were also paralysed, but on the left they retained very slight power. The triceps, pectoral muscles, and serratus magnus could be felt to contract, but only very feebly so. The deltoid, biceps, and supinator longus were more powerful than any of the other muscles of the upper limb, but were also weak.

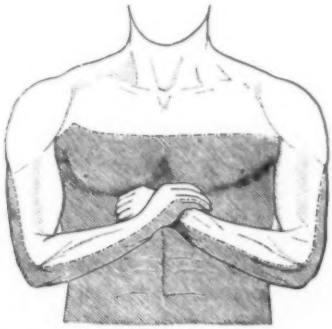

FIG. 2.—Position occupied by the limbs in a case of complete transverse destruction of the spinal cord immediately below the nuclei for the subscapularis muscles.

Both palpebral fissures and pupils were small, the right being more markedly so.

The bladder was much distended, and on passing a catheter a large amount of normal urine was drawn off.

Anæsthesia was very clearly defined, extending over the lower limbs and up the trunk as high as the level of the second rib in front, and to the second dorsal spine and spines of the scapulæ behind. In the upper limbs it affected the little finger and inner part of the ring finger, the ulnar side of the hand and fore-arm, and the intero-posterior aspect of the arm, as shown in the sketch by the shaded portions. The rest of the upper limbs was sensitive. About the boundary of the anæsthetic region was

slight hyper-æsthesia, more marked in some parts than in others.

The penis was semi-erect. Temperature 99°·8.

On the following day the urine was ammoniacal and of a deep purple colour from admixture of blood. This profuse hæmorrhage from the kidneys continued for some days, during which time the patient became gradually weaker, but it ceased on October 10; the urine continuing, however, to be ammoniacal and containing some pus.

On October 9 the temperature in the right auditory meatus was found to be 101°·2 as against 100°·2 on the left side, this observation coinciding with the more marked diminution of the pupil and palpebral fissure on that side due to paralysis of the sympathetic.

On October 20 the optic discs were examined and found to be normal.

On October 25 the right upper limb lay across the chest with the elbow to the side, the hand on the upper part of the sternum and the fingers closed. When asked to move it, he only rotated the humerus outwards, but as he did so the supra-spinatus and infra-spinatus could be felt to contract. He still had very feeble power of flexing the elbow, but neither the biceps nor the supinator longus could be felt to contract, and he had lost all power over the deltoid. All the other muscles of the limbs were absolutely paralysed. On the left side the elbow was also close to the chest, but less flexed, so that the fore-arm was at about a right angle to the upper arm. On this side he had more power of flexing the elbow, and could abduct and adduct as well as rotate the shoulder, there being obvious contraction of the deltoid, biceps, and spinati muscles; he had also some feeble power of pronation. On breathing deeply, the levatores angulorum scapularum could be felt to contract. Anæsthesia was the same as on admission.

On November 1 he was much weaker, and it was found almost impossible to draw off the urine, owing to the catheter continually becoming blocked with stringy mucus. Severe vomiting and hiccough also troubled him greatly, and he refused all food. The temperature throughout the case varied from about 99° to 101°. On November 3 he died exhausted, having survived the accident thirty-one days.

At the *post-mortem* examination, which was made by Dr. Harris, there was found rupture of the disc between the last cervical and first dorsal vertebræ, the parts being very movable, but no fracture was detected, and when the *post mortem* was made there was no displacement. The cord opposite the seat of injury was

almost diffluent, and softening extended throughout almost the whole cervical region and for some short distance below, but the remainder of the dorsal region showed no abnormality. The bladder was much enlarged and its walls thickened: on its mucous membrane was a thick, gritty, yellowish-white membrane which came away as a distinct cast: the peritoneal surface was inflamed and adherent to the sigmoid flexure and omentum. In the kidneys were the appearances of acute parenchymatous nephritis.

Here as in Case 5 we find the cord lesion extending above the site of dislocation before the patient came under observation. As before we have the deltoid, biceps, and supinator fairly powerful. The triceps, pectoral muscles and serratus magnus also retain a fair amount of power and appear to have received about an equal amount of damage, justifying their being placed near together in our table. It will be noticed that the attitude shown in Fig. 2 differs from that of Fig. 1 owing to the adductors (chiefly the pectoralis and subscapularis) of the humerus not being completely paralysed. At the end of three weeks we find on the left side that the supra-spinatus, infra-spinatus, deltoid, biceps, and apparently the supinators, subscapularis, and pronators, retain some power; whereas on the right side the supra-spinatus and infra-spinatus are the most powerful muscles, the flexors of the elbow are not quite paralysed, but all the other muscles are. Again therefore the arrangement confirms that of our table, as far as it goes.

Case 7.—J. S. was admitted to the Manchester Royal Infirmary under the care of Dr. Morgan on February 5, 1887, about 10.30 P.M. He had been at a place of entertainment, and when drunk had fallen from a gallery some ten feet in height, but it was not known how he struck himself.

It was nearly twenty-four hours after his admission when I first saw him, and when he presented the following symptoms. The lower limbs and trunk were completely paralysed, respiration being diaphragmatic. In the upper limbs movements were normal, with the following exceptions:—The intrinsic muscles of the hand were paralysed, the fingers being flexed and their extensors (*i.e.* the interossei and extensor communis digitorum) paralysed, whilst any attempt to completely flex them caused slight extension of the wrist; the wrist was held straight, both its extensors and flexors,

but especially the former, being weak. On the right side the extensors of the wrist were weaker than on the left. The lower limbs were completely anæsthetic, as was the trunk, as high as the level of the second rib in front and the spine of the seventh cervical vertebra behind. In the upper limbs anæsthesia was limited with the greatest accuracy to the inner side of the arm and fore-arm, being bounded in front by a line running vertically from the front of the deltoid opposite the second rib, down the centre of the biceps and fore-arm to the middle of the wrist; similarly behind the boundary was a vertical line down the centre of the triceps and back of the fore-arm to the centre of the wrist. In the hand the limiting line sloped, both in front and behind, from the centre of the wrist to the cleft between the fourth and fifth fingers, the little finger being completely anæsthetic, the others not at all so. There was no trace of hyperæsthesia.

Superficial and tendon reflexes were all absent. Both palpebral fissures were distinctly small, as were the pupils, the latter being as usual especially notable in a dim light. The pupils moreover did not dilate on pinching the skin, but they showed very marked dilatation on each inspiration. Urine was retained. Penis turgid. Pulse 80. Temperature, *mane* 98°, *vesp.* 99°·4.

On the following morning some additional points were noted. He had no pain anywhere, but a sense of stiffness in the upper limbs, and no pain was caused by pressing or jerking the head downwards. The seventh cervical spine was thought to be rather more prominent than usual. Respiration was mainly diaphragmatic, aided by the upper scapular and sterno-mastoid muscles; it was at the rate of 24 per minute, without sense of dyspnœa. He had slight cough and some collection of mucus in the bronchial tubes. The plantar, cremasteric, abdominal, and epigastric reflexes, and the tendon reactions at the ankle, knee, wrist, and elbow, were all absent. The urine had a specific gravity of 1028, and contained neither sugar nor albumin. The pulse was 80, soft, and feeble; sphygmographic tracings taken from the radial and the posterior tibial arteries showed nothing unusual in either vessel. The heart was normal. The skin felt dry and warm, although at this time the axillary temperature was only 96°·2, but the chest and abdomen were moister than the limbs; it presented no redness or trace of soreness.

On the third day he felt himself better, especially as he had been troubled with vomiting, which had now ceased. He had less power over the fingers and wrist, the former being flexed, the latter extended and without power of movement. Dr. J. C. Thresh, the clinical clerk, called my attention to the fact, that the

urine (specific gravity 1031) completely solidified with cold nitric acid, owing to the formation of nitrate of urea. The optic discs were a little hazy, with slight venous congestion.

On February 10 the discs were again examined by Dr. Little, who noted these symptoms, but could not definitely assert that there was any pathological change.

These symptoms continued without any important change until February 11, when it was found that on both sides the patient had lost the power of rotating the wrist or extending the elbow, and that no contraction could be felt in the triceps muscles. The fingers and wrists were flexed and completely paralysed. The pectoralis major on either side was also now paralysed, but the latissimus dorsi, teres major, and subscapularis could be felt to contract on voluntary effort. The position of rest of the upper limbs was now as follows: the humerus abducted almost to a right angle and slightly rotated inwards, the elbows fully flexed, the wrists prone, and the hands across the chest with the wrists and fingers flexed. Breathing was much more difficult: he presented some signs of mental hebetude: the whole body was dry and cold, and the urine was still loaded with urea and phosphates (in solution), having a specific gravity of 1033.

On February 12 the subscapularis could no longer be felt to contract and the latissimi were weaker, the teres major remaining about the same as the day before. The humerus was now rotated *externally* more readily than *internally*, thus differing from the condition of the previous day, and it was a greater effort to bring it towards the side. A little redness had appeared over the malleoli.

On February 14 we found that the boundary of absolute anæsthesia was the same as on admission, but some dulness of sensation extended further outwards on the limb. The motor symptoms were unaltered. The optic discs had become more indistinct than before, and the small vessels were quite obscured. Dr. Little concluded that there was now definite optic neuritis.

During the evening of this day he vomited freely both food and "coffee grounds," and, becoming delirious, died about 1 A.M. on February 15, the tenth day after the accident.

The annexed chart (fig. 3, p. 308) shows the very low temperature which was maintained during the entire duration of the case.

At the *post-mortem* examination we found a fracture of the anterior and upper part of the body of the first dorsal vertebra, a small portion being clipped off and remaining attached to the disc above. The seventh cervical vertebra was displaced forwards, carrying with it the above-mentioned fragment of the first dorsal.

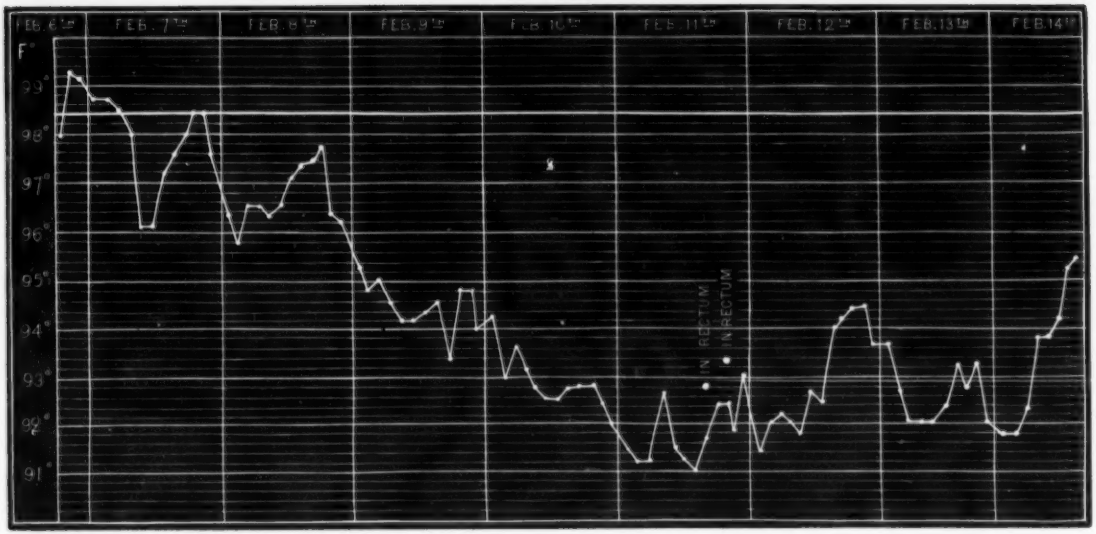

FIG. 3.—Temp rature-Chart in a case of Fracture-dislocation of the 7th cervical vertebra. The temperatures were registered every two hours. At 12 2, 4, &c., till Feb. 9th; at 1, 3, 5, &c. till the 13th at noon; then every three hours. The thermometer was carefully tested and found to be practically correct.

The laminæ of these two vertebræ were separated behind, the articular processes of the first dorsal being posterior instead of anterior to their fellows of the seventh cervical. On opening the spinal canal there was found slight effusion of blood between the bone and the dura mater. The cord was compressed at the level of the first dorsal vertebra, and softened for a short distance above and below the site of compression, its centre being occupied by an

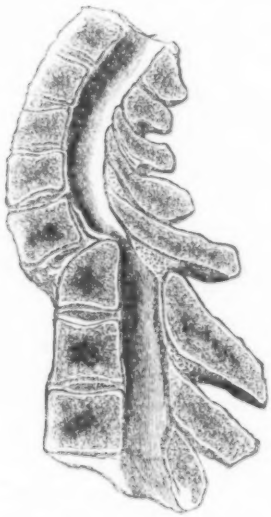

Fig. 4.—Sketch of section of spine in case of J. S., showing fracture-dislocation of the seventh cervical vertebra. From a drawing made by Mr. Moss.

effusion of blood reaching as high as the fifth cervical nerve-roots, in the form of a narrow cone. There was some suppuration between the anterior common ligament of the spine and the fifth cervical vertebra, and at exactly corresponding points on the anterior and posterior walls of the pharynx.

This interesting case is in itself almost a demonstration of the greater part of our table. The injury was quite at the lower part of the dorsal region, and must have at first involved the first dorsal nerves and probably to some extent the eighth cervical. At first we had paralysis only of the intrinsic muscles of the hand and the interossei, but thereafter from day

to day we were able to watch the extension upwards of myelitis, picking out muscle by muscle in the following order:—

1. The flexors of the wrist.
2. The extensors of the wrist.
3. The triceps and pectoralis major.
4. The latissimus dorsi.
5. The teres major and subscapularis.
6. The deltoids, flexors of the elbow, and external rotators of the humerus (supra- and infra-spinati) remained until the last.

The dates at which the pronators failed could not be obtained.

We have thus described the seven cases of fracture-dislocation, and shown that in the main they strongly support the above arrangement. It is true that there are some discrepancies, and some gaps still remain to be filled up, notably with respect to the exact position of the nuclei for the pronator muscles; but considering the nature of the investigation, I think I may fairly claim to have established the original proposition, and may hope with some further observations to verify and complete the results. At least I hope that I have demonstrated the interest and importance of a more exact study of the distribution of paralysis in these cases than is usually attempted—an importance which may be especially great in medico-legal investigations.

In order to illustrate the way in which such observations may assist us to arrive at an exact diagnosis, I now bring forward three more complicated cases of spinal injury, and shall endeavour in each to exactly localise the lesion. A similar accurate study would probably clear up and establish a definite diagnosis in many cases now vaguely described as "concussion of the spine."

Case 8.—R. M., aged 55, a labourer, was admitted to the Manchester Royal Infirmary on April 27, 1887, under the care of Mr. Whitehead. He informed us that about 10 or 11 P.M. on the previous night he had fallen head first down some seven or eight steps, and being able to move his right arm only he had lain as he fell until 7 A.M. I only heard of his admission about 12.30 P.M.,

and then went at once to the ward with Mr. Collier, then resident surgical officer, and Mr. Iddon, house surgeon. When I first saw him, he presented the following symptoms:—The lower limbs appeared to be quite paralysed, but were said to have been moved once or twice during the morning: they were very rigid: the knee-jerk was much exaggerated and the cremasteric reflexes absent. On respiration costal movement was slight, but quite distinct.

Anæsthesia extended as high on the trunk as the second rib, but was not quite complete. Urine was retained; the penis was not turgid. The condition of the upper limbs was more interesting. The left was completely paralysed, lying by the side. The right was abducted at the shoulder, with the elbow strongly flexed, so that the hand lay above the shoulder with the palm backwards, *i.e.* in a state of pronation, the fingers and wrist being slightly flexed. He could raise and rotate the shoulders, but had no other power of movement in the limb. There was almost absolute anæsthesia of the left upper limb, but on the right side loss of sensation was limited to the ulnar aspect. Both pupils and palpebral fissures were small. The head and neck were markedly bent to the right, and there was a distinctly felt lateral curvature in the cervical region. We now decided that there was a dislocation, probably unilateral, of the fifth cervical vertebra, and, having in view the otherwise inevitably fatal termination of the case, we resolved to attempt immediate reduction. This we did as follows:—The man lying on his back in bed, I seized him firmly by the shoulders, and Mr. Collier grasping the head drew it upwards and to the right, then bringing it over to the left. A slight snap was felt, and the position of the head was rendered more nearly but not yet quite straight. The same proceeding being repeated, a snap was again felt, and the head came into its normal position. At this moment the patient screamed with pain shooting down the left arm, and situated, he said, on the side further from the thumb. This pain lasted only for a minute or two. Within half a minute he flexed his left elbow, and then again kept the limb quiet with the elbow flexed. On again examining the sensation of the limbs, we found partial sensibility on the ulnar side of the left upper, but the radial side remained as before. At the same time we noticed a diffused swelling in the left posterior triangle of the neck, due doubtless to effused blood, which swelling I may at once say gradually subsided without giving any trouble. Finally we adapted to the patient's head, neck, and shoulders, a felt casing, designed to keep them in position. No deformity of the head or neck remained. At 2 P.M., an hour later, the patient could move the elbow and fingers on the left side, and sensation

as before was absent only on the radial side of the limb. There was no marked rise of temperature either now or at any subsequent period in the course of the case.

On the following day he passed a large amount of urine, the catheter was not required, and from this time he had no trouble with retention. The urine had a sp. gr. of 1024, contained some albumin and decolorised Fehling's solution, without giving a precipitate; it also contained a large quantity of water. The penis was slightly turgid. There was a good deal of pain between the shoulders, but no other change.

On April 29, the third day, sensation was better in the lower limbs, especially on the right side and in the anæsthetic portions of the upper limbs. The patellar reflexes were less exaggerated than before, and the limbs less rigid. The right upper limb presented as before the symptoms of complete paralysis of motion and sensation below the fifth cervical root: on the left side he could feebly flex and

FIG. 5.—From a photograph of J. M., showing the position of the limbs in a case when the fifth cervical nerve-root was injured on the left side only.

extend the fingers, wrist, and elbow; could pronate and supinate the wrist, and slightly abduct and adduct the humerus. The urine was now alkaline, very fœtid, and contained pus. The optic discs were normal. A day later he had more power in the left upper limb, and anæsthesia was nowhere complete. On the right side he had gained some power of pronating and of extending the elbow, and the area of sensation was extending. The knee-jerks were about normal; the contraction of the pupils less marked. The above figure, taken from a photograph obtained for me by Mr Sidley at about this period, shows the position of the limbs and indicates the marked abduction of the humerus and flexion of the elbow on the right side. From this point it is unnecessary to give a daily record, but it will suffice to note merely a few of the more important changes. Sensation very gradually returned throughout, being preceded, as I have elsewhere shown to be usual, by a

sense of tingling: the right lower limb recovered more quickly than the left. Traces of albumin and sugar disappeared from the urine within a week, as did the pus and the alkaline reaction. For a long time he had some difficulty in passing urine, but this gradually passed off. The pupils returned to their normal condition in about a month, the right doing so more slowly, and even as lately as January 1888 being smaller than the left. For some days he had troublesome bronchitis and all movement of the ribs ceased, but it then gradually returned.

The knee-jerks had quite disappeared by May 5, and after being absent for some time gradually returned in the exaggerated form of spastic paralysis which they still retain, and which is more marked on the right side. On May 11 he could move the lower limbs about the bed very fairly. In about six months he could walk. In the upper limbs return of power was equally slow, and there is here a good deal of atrophy: this atrophy and weakness is still more marked on the right than on the left side. In August all the muscles of the left upper limb reacted well to the faradic current, whereas on the right side only the deltoid, biceps, and supinator longus did so; so that on that side the "reaction of degeneration" was present below the fifth root.

After the accident the nails of both hands grew in a markedly clubbed form.

Since he left the Infirmary this man has frequently reported himself to me, and in April last I showed him to the Clinical Society of Manchester. When I last saw him in August 1888, he could walk well, but with the usual spastic gait, and complained only of a feeling of numbness in the lower limbs, abdomen, and hands. His upper limbs are a good deal wasted, but he has very fair power throughout and can do light work, having no difficulty even in such movements as are required for lacing his boots or buttoning his clothes.

In this case I think we may assume that there was a dislocation of the fifth cervical vertebra, and that the traction thereby caused had injured the left fifth cervical nerve-roots at their point of exit from the cord, so as to completely paralyse sensation and motion in the distribution of that nerve, but that the cord itself was not completely crushed, having only sustained hæmorrhages into its substance. As shown in the accompanying diagram, the right fifth cervical root would escape—the cord below this region would be affected by partial destruction (which was worse on the right side)—and the left

fifth cervical root would, as already stated, be torn or greatly stretched, so as to suffer temporary total paralysis.

Hence we have on the right side partial paralysis of the upper limb; the deltoid, biceps, and supinator longus escaping, as in the other reported cases, where the fifth root was spared, with retention of sensation on the outer side of the limb supplied by the same nerve. All sensation is lost below this root, and the pupil is smaller in size upon this side of the body than on the other, coinciding with the greater amount of damage done to the cord. This is also evidenced by the fact, that anæsthesia was more marked on the left than on the right side in the

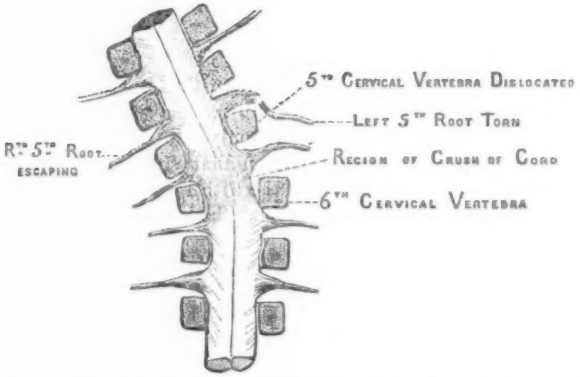

FIG. 6.—Diagram of the supposed nature of the lesion in his case of J. M. The left fifth cervical nerve-root should appear as crushed or stretched rather than completely torn.

lower part of the body, and that the paralysis of those muscles of the upper limb supplied from below the fifth root was more marked on the right side, as was the exaggeration of the tendon reflexes. On the right side also we have the "reaction of degeneration" resulting, whereas on the left it did not do so. On the left side, on the other hand, we find that the movements of reduction, by irritating the overstretched fifth root, caused the muscles supplied by it to contract, but that both motor and sensory paralysis were more marked and enduring in this root than elsewhere. The urinary and other symptoms are compatible with a partial destruction of the cord, and passed off to

a large extent as repair ensued. One point in this case is quite unusual; namely, the early exaggeration of the patellar reflexes, these being as a rule lost, owing to the shock of the injury: whereas here, doubtless from irritation, they were increased;[1] as the irritation subsided, they disappeared, and then at a later stage reappeared in an exaggerated form—the usual result of an old transverse lesion.

Case 9.—J. A., a bailiff, aged 62, attended as an out-patient under the care of Dr. Ross in December 1886. He told us that nine weeks previously he had fallen about nine or ten feet, and, having held out his arms to save himself, came down upon his hands. For a second or two he felt nothing, but had then great pain shooting down the arms to the hands. His upper limbs then became spasmodically contracted, so that he held them with the elbows to the side and flexed, the hands in front and partially flexed —*i.e.* as he showed us, very much in the position in which a book might be held for reading. This pain and spasm lasted for about a fortnight, since which time they have been gradually abating, and he has now not much pain, and can move the arms in all directions, although they are still weak and he cannot well throw them backwards. The legs have felt cold and weak since the accident.

On examination we found hyperæsthesia, cutaneous and deep, extending across the shoulders from the sixth or seventh cervical spine to the acromia. He had no spinal pain or tenderness. On carrying the arms backwards there was pain which prevented his dressing himself. He had no trace of anæsthesia. Over the hyperæsthetic region was a slight herpetic eruption. All the arm muscles appeared to be fairly strong. The lower limbs felt weak, and the knee-jerk was increased. There were no urinary troubles.

On Jan. 18, 1887, I again examined him more carefully. No local signs of spinal injury could be detected. The hyperæsthetic band was limited above at the level of the sixth cervical vertebra, and below by a line some two inches lower; it extended outwards on either side for about three inches—*i.e.* as far as the distribution of the descending branches of the cervical plexus. Its boundaries were well marked, especially above. The herpetic eruption had almost disappeared, and seemed on inquiry to have been due to previous friction with acetic acid.

On examining the upper limbs, I found fair power in the deltoid, flexors, and extensor of the fore-arm and extensors of the wrist,

[1] Since writing the above, I have seen this result in another case.

but marked weakness of the flexors of the wrist and fingers. In the pectorals I found nothing unusual. The hands and flexors of the wrists were wasted to some extent. All the muscles of the upper limbs reacted to moderately powerful faradic currents. No sensory change could be made out in the upper limbs, and there was no jerk on striking the triceps or wrist tendons.

Examined in a dim light, the pupils were distinctly smaller than normal, the change being more marked on the right side, and they did not dilate on pinching the neck. The legs were well developed and presented no paralysis, anæsthesia, or trophic lesions; but the patient stated that they did not feel so strong as formerly, and were always either cold or burning hot. The knee-jerks were markedly exaggerated; the superficial reflexes absent. The bladder and rectum presented no abnormality.

I saw this patient again on April 28, 1888, when he told me that he was much better than he had been at first; but that he still felt some feebleness of the arms and legs, and that occasionally the legs felt hot or cold. He presented no very marked signs of injury, being quite able to go about and perform all the usual movements with his arms and legs. The knee-jerks were slightly, but not much exaggerated. He had no pain, anæsthesia, hyperæsthesia, or cutaneous eruption. His arms were, however, not very strong, and there was obvious wasting of both triceps muscles. On making an electrical examination, I found that the flexors of the fore-arm required a more powerful faradic current than the biceps, the extensors a still more powerful one, and the triceps could only be got to contract with very strong faradic currents. With the galvanic current cathodal closure in every muscle caused contractions more readily than anodal closure, so that there was no reaction of degeneration. Hence the muscles of the upper limb were partially atrophied and insensitive to the faradic current in varying degrees, the most healthy being the biceps, then the flexors of the wrist, then the extensors, and the weakest being the triceps.

This case would appear to be one of hæmorrhage into the central grey matter of the spinal cord, most severe in the neighbourhood of the triceps nuclei, but extending into those immediately above and below. The flexors of the elbow and the shoulder muscles escaped entirely the destroying lesion, but irritation caused the spasmodic position of the limbs which he at first experienced; similarly there was spasm below the region of the hæmorrhage causing flexion of the fingers and wrist. Hence we appear to have had spasm in

two groups of muscles—those above and those below the lesion: viz. above, the biceps, subscapularis, teres major, latissimus dorsi, and pectoralis major; and below, the flexors of the hand and fingers. At the stage when we saw the man this spasm had passed away: the paralytic symptoms were most prominent, affecting the central group of muscles—the triceps, and to a rather less extent the extensors of the wrist and fingers. The region of hyperæsthesia corresponded to the posterior divisions of the central nerves of the plexus. It is notable that we found no sensory affection in the distribution of the anterior divisions, *i.e.* in the central part of the arms, but from various observations I am inclined to believe that the central nerves of the brachial plexus have a very slight distribution in the upper limbs, and doubtless the anterior and posterior bands which they do supply would shortly be vicariously supplied by the upper and lower branches on either side of them. The diagnosis of a central hæmorrhage rather than a meningeal lesion must rest chiefly on the rapid onset of the symptoms. Meningitis could not cause symptoms within a few minutes, and a meningeal hæmorrhage would be less distinctly localised.

Case 10.—R. P., aged 25, a porter, was admitted to the Manchester Royal Infirmary under Mr. Hardie's care on December 19, 1887. He had on the same day been walking along the streets, which were slippery, when he fell at full length on his back. He was seized with a sudden pain at the lower part of the neck, shooting down the arms, and at once sustained complete loss of power in his legs and partial loss in his arms. He was never unconscious.

When examined, he presented severe pain in the lower cervical region, shooting thence down the arms. No deformity of the spine could be felt, but pressure caused great pain between the fourth cervical and first dorsal vertebræ, and pain was caused by pressing the head downwards or moving it from side to side. He lay in bed with the fore-arms folded across the chest, the elbows being a little abducted, the hands prone; abduction and adduction of the humerus were however fairly well performed, as were pronation and supination. Flexion of the fore-arm was not much impeded, but the power of extension was lost. The grasp of his hand was exceedingly feeble, and extension at the wrist resulted from the effort. The flexors and extensors of the wrist were weakened, apparently about equally so. The intrinsic muscles of the hands

were very feeble. These symptoms were similar on both sides, except that on the right adduction of the humerus was less powerful than on the left, whereas the grasp of the hand was stronger on the right side, and unaccompanied by extension at the wrist. On the left side sensation was thought to be somewhat deficient in the fingers, but there was no other affection; on the right side it was normal.

The chest movements were feeble, and the legs showed great loss of power, but sensation was unaffected. The knee-jerk and plantar reflexes were exaggerated on both sides. He had no bladder or rectal troubles; the urine was of sp. gr. 1019, acid, containing a trace of albumin, but no sugar. Temperature, 100° 6; pulse 80. Ext. ergot. liq. was administered several times.

Two days later the arms were slightly stronger, the legs decidedly so; the temperature had come down and shortly reached the normal.

On December 27 extension of the fore-arms was still weak, but the other movements much better. He had now ceased to retain the fixed position described above, except at times, and had hardly any pain. Improvement was rapid, and on January 16, 1888, he was discharged cured, except for some slight feebleness of the grasp.

The above account is taken from notes by Mr. Bannister, the house surgeon, as I only saw the case a few days before his discharge.

This is another instance of a small hæmorrhage, mainly in the region of the triceps nucleus—a hæmorrhage confined to the anterior cornua, and affecting only the motor apparatus. It is needless to repeat the evidence, which shows that it was most severe in the region of the triceps nucleus, but extended thence to those of the extensors and flexors of the fingers, and that it was at a rather higher level on the right than on the left side, so that on the former the abductors of the humerus were also weakened.

The distribution of the *anæsthesia* in the above cases is of interest, equally as great as that of the paralysis. In his most interesting and important paper in the January number of 'Brain,' Dr. Ross has fully demonstrated the nature of the distribution of the sensory nerves of the upper limb. Regarding the limb in its embryological position, we find that it is projected as a bud from the trunk, the hand being supine and the radius upwards, so that the palmar surface is

anterior. In this position the bud carries out with it branches of the anterior primary divisions of the spinal segmental nerves from the fifth cervical to the first dorsal inclusive; and as those nerves maintain in the adult their embryological relations, we have the several roots supplying the limb in numerical order from the radial to the ulnar side. Hence, then, the higher the paralysis extends in the cord through the brachial region, the further will the anæsthesia extend from the ulnar towards the radial side. This arrangement is fully demonstrated by our cases. It is unnecessary to refer again to the distribution which will be obvious to any one who takes the trouble to read the reports, and of which I have already spoken in my previous paper. It will be found that the fifth root supplies the region overlying the deltoid muscle, and the outer aspect of the arm and fore-arm as far as the styloid process of the radius or base of the thumb, and that the eighth cervical and first dorsal supply the little finger and inner side of the hand, fore-arm and arm, the remaining roots providing for the central parts of the limb on both anterior and posterior aspects. It will, however, be found that these central roots supply relatively a much less extensive area than those above and below them. Thus in Case 7 the extension of the anæsthesia was much slower than that of the paralysis, and in Case 9, where the lesion affected the centre of the brachial region, this sensory disturbance was at the end of nine weeks appreciable only in the posterior primary divisions, there being none in the limbs. In Case 10 there was no definite anæsthesia, owing probably to the hæmorrhage being localised to the anterior cornua; the region in which, as I have before shown, it is always most marked.

In referring to certain other points in these injuries, I shall consider the whole of my nineteen cases together.

As regards the *nature* and *seat* of the lesion we have thirteen cases of fracture or dislocation and six of hæmato-myelia, or possibly five of hæmato-myelia and one of meningeal hæmorrhage. The dislocations were thus distributed:—

Between the	4th	and	5th	cervical vert.	3	cases.
„	„	5th	„	6th „ „	5	„
„	„	6th	„	7th „ „	1	case.
„	„	7th	„	1st dorsal	2	cases.

In Cases 1 and 2 of the present paper there were no *post-mortem* examinations, but probably the injury was a fracture dislocation of the fifth vertebra. All the cases conform to the usual type, the upper vertebra being dislocated forwards. In every case which I have examined there was some fracture accompanying the dislocation; the usual condition being either a distinct fracture, or a mere "grinding off" of the anterior superior edge of the lower vertebra. Further injury to the bones may or may not coexist. In two cases which I did not see there was said to be no fracture, but the coincidence is so remarkable that I am inclined to suspect that the above described "grinding off" was unnoted. This fracture is often so slight that, unless a vertical section be made through the affected bones, it can hardly be recognised. The condition is represented in Fig. IV. where such a section was made. In Case 4 only the dislocation was lateral, but as the patient is living the exact condition cannot be ascertained. In the three cases of hæmato-myelia previously reported, the hæmorrhage was situated about the level of the fourth or fifth root, the region which, as I then explained, is most liable to this injury. Of the two cases here given, both were about the sixth root.

It is not always easy to ascertain the exact action of the force which *caused* the injury, but of the nineteen cases probably nine or ten were due to over-extension or over-flexion of the neck (Nos. I., II., VII., 1, 2, 3, 4, 5, 6, and 8).[1] In five cases (III., IV., VI., VII., and 10) a blow or crush of the shoulders was the source of the injury. One (IX.) was a gunshot wound. One (9) was due to a fall upon the outstretched hands, and may therefore probably be regarded as due to jerking of the head forwards. In two the nature of the injury was very obscure—they were both falls, and there was nothing to show how the patient struck himself.

In only five cases was there noted any *local deformity*, and in many of the others its absence was remarked upon: in this connection it must however be remembered that I only saw these cases after they had been laid flat in bed—a procedure

[1] I refer to cases in the earlier paper by Roman numerals, to those in the present one by Arabic numerals.

which frequently reduces the deformity. In dislocations *local pain* is invariable. As it is frequently suggested in courts of justice that a fracture or dislocation of the spine is always accompanied by pain on pressing downwards upon the head, I have recently paid particular attention to this point; and in five cases of dislocation and two of hæmato-myelia where I sought for it at an early period, I found the symptom once only, and that in a case of hæmato-myelia (10).

Hyperæsthesia is a symptom much less commonly found than the text-books would lead us to suppose. Among the fracture dislocations it was only once (Case 5) distinct, and twice (Cases 4 and 6) was slightly marked; in all the others it was absent. In Case IX., a gunshot wound, with probably meningeal hæmorrhage, it was well marked; and in Case 9, one of hæmato-myelia, it was also very distinct. Hence I can see no value in this presence or absence of hyperæsthesia, which is commonly stated to be a valuable diagnostic sign between dislocations and fractures respectively.

The *tendon reactions* were noted at an early period in eight cases of fracture-dislocation, in seven of which they were lost immediately after the accident. In one case (8) they were markedly exaggerated. The loss of these reactions is attributable to the shock of the accident, and in the exceptional cases we must assume that the lesion caused great irritation of those portions of the cord situated below it. In this instance they shortly disappeared and were lost on the eighth day. I had wished to ascertain the period at which the knee-jerks return, but as most of the cases die too early this is not easy to do. In Case 4 return was between the fourteenth and nineteenth days. In Case 8 they were still absent at the end of the seventh week, but I have no further note until the fifteenth week, when they were again very much exaggerated, my note at the time being "the most marked I have ever seen." In Case I. there was no return at the twenty-first day—the date of death—and all the other dislocations died before return was possible. Of the cases of hæmato-myelia we find in VII. exaggeration on the eleventh day, and in all it was found sooner or later; in Case 10 it was noticed immediately. Hence then we can lay down no definite rule as to the

period at which spastic symptoms ensue, having every variation from an immediate onset to one delayed for over seven weeks.[1]

The *superficial reflexes* are generally lost below the seat of the lesion. Their condition was noted in eleven cases. In ten they were lost immediately or very soon after the accident. In Case 5 (a woman) the plantar reflex was exaggerated and the abdominal present, but as the patient only survived for forty hours, we cannot tell how long they would have remained so: it is notable that this is the only case with marked hyperæsthesia.

Contraction of the *pupils* and *palpebral* fissures has been already sufficiently dwelt upon: all of these cases illustrate the condition. We have, however, to refer to one point not hitherto noted, namely, the fact that the pupil does not dilate on irritating the skin; the physiological explanation is obvious. I have been anxious to ascertain the condition of the optic discs, and have examined the majority of the cases, although I only find notes in four. In those which were examined, but where there is no note, the condition was certainly normal. In Cases 6 and 8 no change could be detected—during periods sufficiently remote from the accident. In none of the cases which recovered was there any complaint of eye symptoms, so that even without an ophthalmoscopic examination we may assume that there was no optic neuritis. In two cases (4 and 7), however, we find very distinct changes. In Case 4 the discs were on the fifth day ill defined and showed venous congestion, but no further changes occurred during life (twenty-eight days). In Case 6 there was similar haziness and congestion on the third day, and distinct neuritis on the ninth; as death occurred on the tenth, the results could not be followed. I am aware that on such a point the ophthalmoscopic observations of anyone but a specialist are open to doubt, but in the present instances I had the advantage not only of seeing the change occur from the healthy to the diseased condition, but also of having my conclusions

[1] I have since seen two cases: one a fracture-dislocation of the cervical region, when exaggerated knee-jerks appeared at once and lasted until death; another of the mid-dorsal region, where there is still no knee-jerk at an interval of six months after the injury.

confirmed by Dr. Little, who kindly examined the cases for me and whose name leaves no room for doubt.

Some interesting *vaso-motor* effects were observed. In twelve cases of fracture occurring in males priapism was present from the first in eleven and persisted until death. In the twelfth case (8) it only appeared on the second day, and gradually passed off as recovery ensued. The thirteenth case of fracture (5) was a woman, and here I was told by the nurse that there was a profuse vaginal discharge: unfortunately this information only reached me after death, and I was unable to examine the nature of the discharge, but it is at least probable that it was a secretion due to changes analogous to those causing priapism in the male—a condition I do not remember to have hitherto seen noted in any spinal affection. On the other hand, of five cases of hæmato-myelia occurring in males, only one presented any turgidity of the penis, and this one only for about twenty-four hours (Case VI.). This I believe to be due to the vaso-motor fibres being situated near the periphery of the cord, and thus escaping the effects of a central hæmorrhage.

The *skin* was generally dry and warm, even when the temperature was low, but there are several exceptions to this condition. In Case 6, where the right cervical sympathetic was more affected than the left (as indicated by the smaller size of the pupil), the temperature was one degree higher in the right than in the left auditory meatus.

High temperatures were only found in four cases (III., IV., V., and 2), all of which were fracture dislocations high up in the brachial region, and all of which died within forty-eight hours. A remarkably low temperature was observed in Case 8, where the lesion was between the last cervical and first dorsal vertebræ.

The *pulse* was usually slow and almost always full and soft, and in Case 3 presented a very remarkable respiratory rhythm; there being an exaggeration of the normally larger amount of blood expelled by the heart during inspiration.

The *urine* was retained in all the cases of fracture dislocation. In all cases of hæmato-myelia it was either not retained at all (VIII., 9 and 10) or only for a short period (in

VI. for one day, in VII. for four days); whereas in IX., a female with meningeal hæmorrhage, there was prolonged incontinence. This I believe to be due to the same reason as the turgidity of the penis, viz. to the fibres for the bladder being peripherally situated. (See above, and also p. 540 of 'BRAIN' for January 1887.)

Transient *glycosuria* was noticed in two cases (5 and 8), and transient *albuminuria* in two cases (8 and 10); increased density of the urine with excess of urea in Case 7; increased secretion of uric acid in Case 8; and of phosphates in several cases. These results, which are by no means constant and which only persist during a few days, would appear to point to important vascular changes in the kidneys.

In Case 6 there was on the second day excessive hæmaturia, with fœtor of the urine—an instance, apparently, of acute trophic lesion of the kidneys. And it is perhaps worth noting that in this case the bladder had not been relieved for two days, and that the hæmaturia came on shortly after the first use of the catheter.

THE PATHOLOGICAL ANATOMY OF A CASE OF LOCOMOTOR ATAXY, WITH SPECIAL REFERENCE TO ASCENDING DEGENERATIONS IN THE SPINAL CORD AND MEDULLA OBLONGATA.

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AND

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THE patient was a male, aged 35, a bricklayer, who was admitted into St. Thomas's Hospital on May 20th, 1886. He was under the care of Dr. Ord, who has kindly allowed us to use the case.

There are many points in the clinical history of much interest, but for our present purpose a brief account of the condition during life is all that is necessary.

The patient stated that his symptoms began two years previously with weakness of the lower limbs and shooting-pains, and that a little later he found difficulty in walking in the dark. About a year after the first onset of the symptoms the patient began to lose control over his bladder, and five months subsequently, after a severe headache, he had double vision and squint. There was no history of syphilis.

On admission, he could not stand or walk without assistance. There was great inco-ordination of the legs, and some loss of power, chiefly affecting the quadriceps femoris.

There was no muscular wasting. Sensation was much impaired in both feet and legs, but elsewhere on the body it was good. The knee-jerks and the tendon reactions of the arms were absent, as were also the plantar and cremasteric reflexes. The abdominal and interscapular reflexes were said to be present, though the latter was slight. There was no loss of power in the arms and no obvious inco-ordination.

The pupils were small, but the left was larger than the right. On admission, it was noted that they acted to light and during accommodation, but after he had been in the hospital six months the pupils were said to exhibit the typical Argyll-Robertson condition.

Three months later, a week before death, it was stated that there was no reaction either to light or during efforts at accommodation. Occasionally he had diplopia on looking to the extreme right, and it was thought that there was slight weakness of the right external rectus. There was no colour-blindness, and the optic discs were normal.

The condition of the patient until death may be summed up in a few words. He had frequent attacks of pain above the symphysis pubis, sometimes accompanied by rigors and elevation of temperature, and usually by an alkaline state of the urine. Several times it was noted that the urine contained phosphates and pus.

Within a few weeks of his death he had on two occasions a series of epileptiform attacks, probably of uræmic origin. During the last week he had hallucinations and was very noisy. He died on February 23rd, 1887.

Post-mortem Examination was made twenty-two hours after death.—Body fairly nourished. There was no urate of soda in the metatarso-phalangeal joints of great toes; but there was some erosion of the cartilages, especially near the margins. No bony outgrowths. Skull-cap and dura mater natural; venous sinuses contained soft clot only.

Brain.—At the vertex the arachnoid was thick and opaque, and the Pacchionian bodies unusually large. There was general excess of sub-arachnoid fluid over the convex surface; the vessels were moderately distended. The pia mater was readily detached. The optic nerves, chiasma and tracts, the third, fourth, sixth and other nerves at the base seemed healthy.

The left vertebral artery was of normal size, the right about one-third the size of its fellow. There was a small localised atheromatous patch on the basilar artery, but with this exception the vessels at the base were healthy.

The motor convolutions were normal. In the cortex of the

left gyrus fornicatus, just in front of the rostrum of the corpus callosum, there was an ill-defined, slightly grey patch, which was possibly an islet of early sclerosis. With this exception there was no sign of degeneration. The central ganglia were healthy. The cerebellum appeared normal.

The bodies of the vertebræ and the dura mater were natural.

Spinal Cord.—Nothing abnormal was seen on the anterior aspect of the cord. On the posterior surface, from the upper dorsal region downwards, the pia-arachnoid was dull-white and thickened, and the sub-arachnoid fluid was in excess. A few small calcareous plates were seen attached to the surface of the arachnoid posteriorly. There were no adhesions between the dura mater and the pia-arachnoid. The posterior roots and ganglia, especially in the dorsal and lumbar regions, were much atrophied, grey and translucent. The anterior roots were natural. The cord was somewhat smaller than normal, but not markedly flattened in front or behind.

Only a few sections of the cord were made in the fresh state. In the lumbar and lower dorsal regions the entire posterior column had a grey, translucent appearance, the posterior horns being also involved. In the cervical region the columns of Goll (postero-median) only seemed affected, but the posterior cornua were very slender.

In the medulla oblongata the fasciculus gracilis and apparently the fasciculus cuneatus on both sides were sclerosed.

The vagi, the right anterior crural nerve, and the right-quadriceps muscle appeared healthy.

Larynx, trachea and bronchi contained some blood-stained mucus.

The following were healthy: the lungs, heart, liver, spleen, adrenals, œsophagus, stomach and intestines.

The first part of the aorta was much thickened generally, and exhibited many large patches of atheroma, but no calcareous plates. In the rest of the aorta there were scattered, raised patches of atheroma.

The urethra was healthy. The bladder was much dilated and hypertrophied. Both lateral lobes of the prostate were softened, of a dirty-slate colour, and infiltrated with pus. The ureters, which were dilated to two or three times the normal size,

contained some turbid urine. The pelves and calyces were much dilated, a little injected, and contained a few small concretions of uric acid, but no pus. The capsules of the kidneys were much thickened and adherent to the surrounding tissues. The capsules were readily detached; but the surface of the kidneys was irregular and lobulated, and showed some small, round points which were probably minute abscesses. On section, the cortex was irregular and very obscure, but exhibited no suppurative points.

The spinal cord, medulla oblongata and pons were hardened in Müller's fluid and subsequently placed in spirit. In the spinal cord sections were made at the level of each nerve-root from the apex of the conus medullaris upwards. The staining was by the Weigert hæmatoxylin process.

Microscopical Examination. Spinal Cord: at the level of the coccygeal nerve.—Even here, the lowest part of the cord, the posterior columns are much sclerosed. Some of the nerve-fibres on each side of the postero-median fissure still remain. The area of fine nerve-fibres lying in front of the entrance of the posterior nerve-roots (Lissauer's tract) is but little degenerated. The network of fine medullated fibres has disappeared from the posterior horns, and no nerve-fibres can be seen passing from the posterior root-zones to the cornua, as occurs in the normal state. There is an evident excess of fibrous tissue in the lateral columns, especially just in front of the posterior cornua.

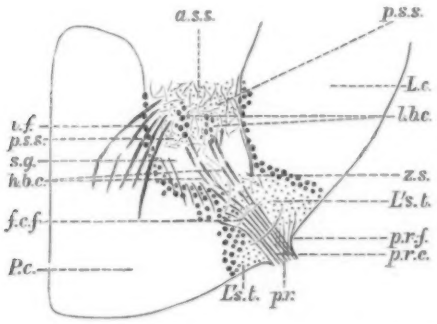
Sacral region.—The posterior sclerosis is well marked; but on passing from the lower to the upper part of the sacral region a gradual increase in the number of healthy nerve-fibres is seen in that part of the posterior columns which abuts on the posterior grey commissure. This tract of normal fibres extends backwards as a wing-like expansion, following the contour of the cervix cornu posterioris on both sides. The horizontal fibres which normally pass from the postero-external columns to the posterior cornua are almost absent. In the caput cornu posterioris the substantia spongiosa, especially in its posterior part, contains far fewer medullated fibres than it does normally. On the other hand, the reticulate region of the grey matter immediately in front of the posterior cornu, is quite healthy.

At the level of the third sacral nerve, Lissauer's tract is profoundly degenerated.[1]

(Figs. 1 and 2 represent the gross characters of the lesion in the lower and upper sacral regions.)

Lumbar region.—The degeneration of the posterior columns is greater here than in any part of the sacral region. In the postero-external columns, behind the cervix cornu posterioris, there are two symmetrical areas of fairly healthy nerve-fibres. Throughout the lumbar region, Lissauer's tract is completely sclerosed, and the horizontal radiations from the posterior root-zones into the cornua are absent. In the lateral columns there is diffuse sclerosis, the degeneration being much less advanced than in the posterior columns. The lateral sclerosis does not merely include the pyramidal-tract area, but extends far beyond it, especially anteriorly and towards the middle line.

[1] We append a Schema of the constitution of the posterior cornu in the lumbar region according to Lissauer (slightly modified from the original in the 'Archiv f. Psychiatrie,' vol. xvii. p. 377).



SCHEMA OF THE CONSTITUTION OF THE POSTERIOR CORNU, ACCORDING TO LISSAUER.

L.c., lateral column.
P.c., posterior column.
p.r., posterior nerve-root.
p.r.c., coarse fibres in *p.r.*
p.r.f., fine fibres in *p.r.*
L's.t., tract of Lissauer.
a.s.s., anterior region of substantia spongiosa.
p.s.s., posterior region of substantia spongiosa.
s.g., substantia gelatinosa.
z.s., zona spongiosa.
l.b.c., longitudinal bundles of large fibres.
h.b.c., horizontal bundles of large fibres.
f.c.f., fine commissural fibres.
i.f., irradiating fibres from posterior column.

Dorsal region.—Between the ninth and tenth dorsal nerves the sclerosis in the posterior columns reaches its greatest intensity. The few fibres that remain occupy an area on each side behind the cervix cornu posterioris. There is great diminution in the number of the medullated fibres in the posterior cornua, and also in the posterior vesicular columns, except just in the front of the cell-groups. The fibres which are mainly affected in Clarke's columns are those situated internal to the cells and which pass to the posterior white commissure. There is no obvious alteration in the appearance of the cells in the posterior vesicular columns; but after a careful comparison with healthy sections the large nerve-cells seem fewer than normal, the small cells presenting no diminution in number.

In the substantia spongiosa of the posterior cornua the horizontal fibres are quite absent, whereas there are a considerable number of fibres running vertically. The medullated fibres of the posterior commissure have suffered severely. The horizontal radiations from the posterior root-zones and the horizontal fibres in the substantia spongiosa of the posterior horns are absent.

. The formatio reticularis is quite healthy. The lateral columns, however, are much sclerosed, and the change, as in the lumbar region, spreads far beyond the pyramidal-tract area and reaches up to the periphery of the cord. About the level of the seventh dorsal pair the change in the lateral column extends forwards, and gradually becomes larger and more defined on passing upwards. At the level of the eighth dorsal nerve-root the posterior vesicular columns and the cells contained in them become more obvious, and the fine vertical nerve-fibres normally surrounding the columns can be distinguished. On the other hand, there is still a marked paucity of horizontal fibres both in Clarke's columns and in the substantia spongiosa of the posterior cornua, and the horizontal radiations of the posterior root-zones seem to be destroyed.

In the lower dorsal region Lissauer's tract is completely degenerated, but in the upper dorsal region it is less affected, and the anterior part seems normal.

In the posterior columns more nerve-fibres are now visible,

and at the level of the sixth dorsal nerve-root they are collected into groups symmetrically placed in the postero-external columns (see Fig. 3). On passing upwards, these symmetrical areas gradually increase in size, and the nerve-fibres contained in them are arranged more compactly. The situation and dimensions of these islets at the level of the third dorsal nerve are represented in Fig. 4, and Fig. 5 exhibits them at the level of the eighth cervical nerve-root.

Cervical region.—Below the level of the eighth cervical nerve the posterior pair of these symmetrical areas is made up of nerve-fibres of large size, but now a small number of fine fibres can be distinguished.

In the lower half of the cervical enlargement the groups rapidly increase in size, and at the level of the sixth cervical nerve the anterior and posterior groups on each side become continuous. This condition of things is well seen at the level of the fifth cervical nerve (Fig. 6). The comparatively healthy area in each posterior column, formed by the fusion of the anterior and posterior groups, is roughly wedge-shaped, and its base, which corresponds with the free surface of the posterior column, passes some distance into the postero-median column. The grey matter above the level of the seventh cervical nerve-root is healthy, and it must be specially noticed that the fine medullated fibres in the posterior cornua show no signs of degeneration. About the middle of the cervical enlargement Lissauer's tract seems normal throughout.

The change in the lateral columns is seen at the periphery of the cord as a well-defined area, which gradually increases in size on passing from the lower to the upper cervical region (compare Figs. 5, 6 and 7).

Medulla Oblongata.—The change in the lateral columns can be followed from the highest cervical region into the medulla, where it occupies the position indicated in Figs. 8 and 9. It is wedge-shaped, with its base directed outwards and extending to the periphery, the apex lying between the antero-lateral nucleus in front and the substantia gelatinosa behind. The deep arciform fibres are not degenerated, and they can be seen coursing circumferentially through the sclerosed area in Fig. 9.

On passing upwards in the medulla the tract becomes less

distinct and lies more posterior, until finally it passes into the restiform body. The difficulty in tracing the tract is due partly to the fact that it is divided obliquely by the ordinary transverse sections, and partly to the fact that it is no longer a compact area, but becomes broken up and scattered among other fibres.

The fasciculi graciles, like the postero-median columns throughout the cord, are densely sclerosed, the degeneration being traceable as high as their nuclei. The fasciculi cuneati are much less completely sclerosed, and here again the change can be followed up to the level of the nuclei.

Remarks.—There are some points of pathological interest in this case to which we wish to direct special attention. The lateral columns exhibit evident degeneration from the coccygeal region upwards, the change in the lumbar and lower dorsal regions being diffuse and extending far beyond the pyramidal-tract area. In the upper dorsal and cervical region the degenerated area becomes more defined, reaches up to the periphery of the cord, occupying a position midway between the anterior and posterior nerve-roots. This sclerosed area, when traced upwards, is found to be directly continuous with the wedge-shaped patch of degeneration in the medulla oblongata, lying in front of the substantia gelatinosa (Fig. 8). Now this is the site of the direct cerebellar tract, and the position usually assigned to this tract in the spinal cord is not degenerated in our case.[1] This being so, it is reasonable to infer that the so-called "antero-lateral tract" in the spinal cord is simply the more ventral or anterior portion of a tract proceeding to the cerebellum, in other words, of the direct cerebellar tract.

We may remark, in passing, that in normal sections this sclerosed area in the medulla oblongata is occupied almost exclusively by nerve-fibres of large diameter, similar to those composing the direct cerebellar tract and the so-called antero-lateral tract; and that, moreover, these large nerve-fibres

[1] On referring to Fig. 6, it will be seen that there is slight degenerative change behind the antero-lateral tract on one side, limited to the periphery. This may be secondary to the thickening of the pia mater. In no other part of the cord was there the slightest suspicion of change in the direct cerebellar tract.

in the medulla become oblique on passing upwards, enter the restiform body and so reach the cerebellum.

With respect to the change in the lateral columns in this case, it is interesting to note that the lesion appeared to bear a relation of an inverse nature to that in the postero-external columns. As the latter became more and more occupied by healthy nerve-fibres, the degenerated areas in the lateral columns become denser and larger. It seems to us very probable that there is a supplementary relation between these two regions of the cord. The anatomical characters in our case suggest that certain fibres of the posterior nerve-roots, after an upward course of longer or shorter extent, pass forward into the lateral column of one or both sides. If this be so, it would appear that some of the fibres of the posterior nerve-roots ascend directly to the cerebellum.

It is difficult to pronounce positively that the change in the lateral columns is of a systemic nature, but the probability is that such is the case. This view is borne out by the condition of things seen in the medulla, where the deep arciform fibres pass quite unaffected through the sclerosed area (see Fig. 9).

It is held by some observers that the antero-lateral tract finally enters the antero-lateral nucleus in the medulla oblongata. This view has been adopted by Dr. Tooth in a recent communication,[1] and in support of this he quotes an observation by Westphal.

Dr. Tooth observes that "the cerebellar element is seen in the restiform body, the antero-lateral remaining where it was: higher still the same tract is very small, evidently close on disappearing. One thing seems certain, viz. that these fibres do not pass to the cerebellum *viâ* the restiform body." Our observation, however, is not in harmony with this view. It is interesting to note, in relation to this point, that in our case the fibres of the degenerated area in the medulla entered the restiform body at a higher level than is usually the case with the direct cerebellar tract.

[1] "Note on the Ascending Antero-lateral Tract."—'St. Bartholomew's Hospital Reports,' vol. xxiii. See also vol. xxi., "A Contribution to the Topographical Anatomy of the Spinal Cord." Dr. Tooth here admits the possibility, in the case narrated, that the antero-lateral tract passed with the direct cerebellar tract into the cerebellum.

Dr. Gaskell has shown that there is a sharp distinction, both anatomical and physiological, between the small and large nerve-fibres in the anterior roots. It would be of the greatest interest to obtain more information respecting the small fibres in the posterior nerve-roots. In our case it will be noted that these fibres, as well as those of the larger kind, are degenerated in the greater part of the cord, and that Lissauer's tract, which is the continuation of these small fibres, is also profoundly affected. From the facts at our command, we are not in a position to afford any information concerning the function of these fine nerve-fibres of the posterior roots.

Our conclusions may be summarised as follows:—(1) The antero-lateral tract passes to the cerebellum through the restiform body, occupying in the medulla the position of the direct cerebellar tract. (2) The antero-lateral tract is the more ventral or anterior part of the direct cerebellar tract. (3) The fibres which compose the antero-lateral tract, at any rate in part, proceed from the posterior spinal nerve-roots, and these latter are thereby brought into direct relation with the cerebellum.

Addendum.

We had originally intended to deal with the subject of ascending degenerations more fully, and with this view Fig. 10 was added to the plate. The case was interesting, both clinically and pathologically; but a brief account is all that is necessary at the present time. In 1876 the patient was in St. Thomas's Hospital, suffering from angular curvature and spasmodic paraplegia. He regained almost complete power, and when he was admitted again in 1883, it was noted that he was "able to walk perfectly." After his death, which occurred a little later, the cord was found profoundly degenerated. Below the point of compression the lateral sclerosis was very marked. Just above, as seen in Fig. 10, there was also change in the lateral columns, but to a less intense degree. The degeneration partially involved the pyramidal-tract area. It may be noted here that the pyramidal-tract area normally contains fibres of different

Fig.1.

Fig.2.

Fig.3.

Fig.4.

Fig.5.

Fig.6.

Fig.8.

Fig.7.

Fig.10.

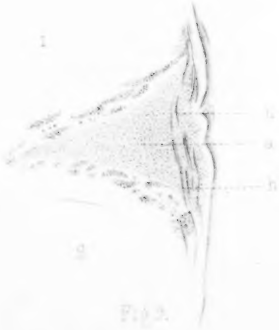


Fig.9.

size and probably of different function, and there is little doubt that certain of these fibres, as seen in the fig., degenerate in an upward direction.

EXPLANATION OF PLATE.

FIG. 1. Transverse Section of Cord in Lower Sacral Region.
FIG. 2. " " " Upper " "
FIG. 3. " " " at 6th Dorsal nerve-root.
FIG. 4. " " " 3rd " "
FIG. 5. " " " 8th Cervical "
FIG. 6. " " " 5th " "
FIG. 7. " " " 2nd " "
FIG. 8. " " Medulla Oblongata opposite the upper pyramidal decussation.
FIG. 9. The degenerated area in Fig. 8 enlarged.

All the figures, except 4 and 9, are twice the size of the mounted specimen, from which they were outlined with the camera lucida. Fig. 4 is magnified ten times, and Fig. 9 thirty times.

a = sclerosis in the lateral column, concentrated and well-defined.
b = diffuse sclerosis in the lateral column.
c and *d* = the anterior and posterior islets of nerve-fibres in the posterior columns.
e = degeneration in fasciculus cuneatus.
f = degeneration of fasciculus gracilis.
g = corpus Rolandi.
h = fibræ arcuatæ profundæ.
i = nucleus antero-lateralis.

FIG. 10 (see Addendum). Dorsal region of the cord above the point of compression in a case of angular curvature (magnified two diameters). The stippled parts indicate the healthy tissue, which in the mounted specimen was coloured dark by the Weigert process.

E F = degenerated posterior column. A = degeneration in lateral column.

ON THE EVOLUTION OF THE CENTRAL NERVOUS SYSTEM OF VERTEBRATA.

BY J. BLAND SUTTON, F.R.C.S.

Hunterian Professor; Assistant-Surgeon, Middlesex Hospital.

In this Journal (January 1888) I attempted to show, from pathological and embryological data, that the intimate relations which exist between the spinal cord and alimentary canal, may be explained on the assumption, that the central canal of the nervous system may be regarded as a modified portion of bowel, and the principal facts adduced in support of that opinion were as follows:—The continuity of the gut and central canal of the cord in the embryo; their similarity in mode of development and correspondence in point of time; the relation of the sympathetic ganglia to the gut and cord; the association of certain malformations of the central canal of the cord with malformations of the alimentary canal; and the relatively large size of the cord in lower vertebrata.

Nearly three years ago I ventured to state, "notwithstanding that we were absolutely in the dark concerning the significance of the central canal of the cord, it was justifiable to regard it as an obsolete canal, basing this opinion on the ground that it behaved itself like functionless ducts and passages in general, and at times formed cysts." [1]

It gave me considerable satisfaction to hear Dr. Gaskell express, before the Anatomical Society of Great Britain (assembled at Cambridge in June 1888), the opinion, that the tube which primarily represents the central nervous system in the vertebrate embryo, must be regarded as a disused segment of the primitive alimentary canal. Dr. Gaskell had arrived at this conclusion from quite a different mode of

[1] "On the Conario-hypophysial Tract," &c.

working to that adopted by me: it is of interest and importance that two men working independently and in different directions should arrive at fundamentally the same conclusion, although, as may be anticipated, there is some difference in minor details.

As far as I am aware, nothing in any way approaching this somewhat startling conclusion has ever been suggested; exception, perhaps, should be made to the unsatisfactory attempt of Owen,[1] who supposed that the gullet of ancestral vertebrates passed through the third ventricle, and that the space from the pineal to the pituitary bodies, by way of the infundibulum, represented this primitive or invertebrate (cephalopodic) gullet.

Invertebrates are distinguished embryologically from the majority of vertebrates by the fact that, in the early stage of development, they undergo a process termed invagination, whereby a portion of the outer wall of cells is depressed in

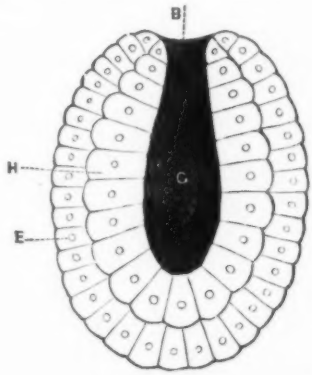


FIG. 1.—A diagram representing the gastrula. E. Ectoderm (epiblast); H. Endoderm (hypoblast); G. Archenteron; B. Blastopore.

such a way as to give rise to a double-walled cup (Fig. 1). This is the gastrula stage, and represents temporarily the double-layered condition permanent in many invertebrata. The hollow, G, in the gastrula is the archenteron, the primitive digestive cavity; the cells lining it constitute the endoderm

[1] 'Journal of the Linnean Society,' 1883, p. 131.

(hypoblast); the outer wall is the ectoderm (epiblast); the two layers become continuous at the edges of the blastopore. It should be mentioned that the gastrula stage occurs in amphioxus as in invertebrata, and has been detected in the ova of some fish and amphibia.

Ingenious attempts have been made to identify the occurrence of something, which in the higher vertebrates corresponds to the gastrula stage of invertebrates, for the intimate connection of the embryological processes throughout the animal kingdom strongly suggests the occurrence of the gastrula in a modified form, even in the highest mammals. Up to the present time nothing satisfactory has been advanced in this direction. The views which I shall now endeavour to express were forced upon me whilst investigating the mode of origin of those singular ovarian tumours known as dermoids.

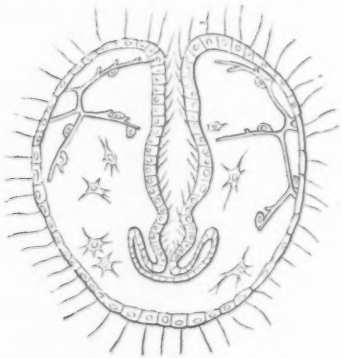


FIG. 2.—Transverse section of the embryo of *Echinus miliaris* to show the origin of the pleuro-peritoneal cavity (cœlom) from the primitive digestive cavity. (From Hertwig, after Selenka.)

It has long been customary to regard the cœlom (pleuro-peritoneal cavity) as a dependency of the vascular or lymph system, but the investigations of the Hertwigs[1] show this view of the matter to be untenable; it is on the contrary a derivative of the archenteron. Each pleuro-peritoneal cavity, for they are at first quite separate, arises as a diverticulum from the central cavity of the archenteron as represented in Fig. 2.

[1] '*Die Cœlomtheorie.*' Jena, 1881.

The central portion persists as the permanent alimentary canal, whilst the lateral diverticula converge ventrally and coalesce to form a common chamber. A transverse section of the embryo of amphioxus is represented in Fig. 3, in which those portions of the primitive digestive cavity which become abstricted to form the body-cavity, are clearly indi-

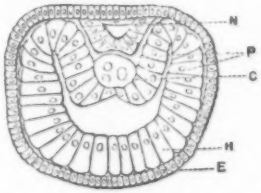


FIG. 3.—Transverse section of the embryo of Amphioxus, showing the abstrictions of the primary alimentary canal which form the cœlom. (After Kowalevsky.) N. Medullary folds; C. Notochord; H. Hypoblast; E. Epiblast; P. Cœlomic epithelium.

cated, whilst Fig. 4, representing a Polygordius larva in transverse section, shows the relation of the diverticula to each other when the abstriction is complete. It may perhaps be more conclusive to indicate the relation of these parts to each other in a vertebrate. In Fig. 5, a transverse section through the head of an embryo dog-fish is represented, and the relation

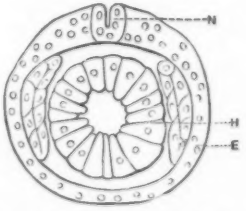


FIG. 4.—Transverse section of a *Polygordius* larva, to show the relation of the alimentary canal and the rudiments of the cœlom. (After Hatschek.) N. Medullary folds; H. Hypoblast; E. Epiblast.

of the medullary folds to the gut and cœlom is clearly shown. This view of the nature of the cœlom is of great importance from a pathological stand-point, because it clearly indicates that the lining of the peritoneal cavity is a true

epithelium of hypoblastic origin. Embryologically it is equally important, as it tends to show that the peculiar manner in which the cœlom arises in most mammals, viz. as a schizocœle, must be regarded as a secondary or modified form of invagina-

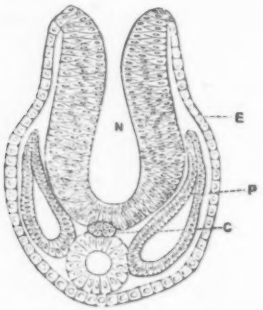


Fig. 5.—Transverse section through the head of an embryo dog-fish, showing the parts representing the gastrula cavity. (After Balfour.) N. Medullary folds; E. Epiblast; P. Cœlom, or pleuro-peritoneal cavity; C. Notochord.

tion, and represents only a part of the gastrula stage of invertebrata.

We may now turn our attention to the space in the gastrula which lie between the blastopore and the wall of the tube we have just been considering. In vertebrata, one of the most

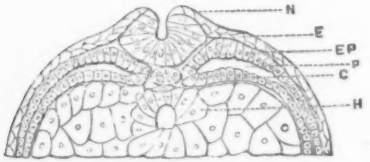


Fig. 6.—Transverse section of a three days' embryo of *Triton tæniatus*, showing the cœlom arising as a schizocele. (After Hertwig.) N. Medullary folds; E. Epiblast; H. Hypoblast; C. Notochord; P. Cœlom.

conspicuous structures in the early embryo is the notochord, concerning which a great deal of discussion has been carried on at various times as to which of the germinal layers it is derived from. Morphologically, its most conspicuous features are its close association with the alimentary canal, and relation

to the spinal cord.[1] If we glance at the gastrula of *Echinus miliaris* we find that by approximating the edges of the archenteron at the point N, we should produce a thickening, and divide the cavity into a dorsal and ventral section, the part below corresponding to the bowel and cœlom, and the parts on the dorsal aspect would represent the medullary folds of the vertebrate; now occlude the blastopore, and we at once give rise to an arrangement of parts corresponding in transverse section to the disposition of parts seen in the early vertebrate embryo, and in longitudinal section would furnish us with the fundamental U-shaped tube from which I started in the previous communication. Such a relation of parts is shown in the longitudinal section of the embryo of the amphibian *Bombinator igneus* (Fig. 7).

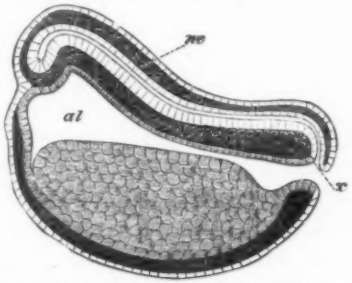


FIG. 7.—Longitudinal section of an embryo of *Bombinator igneus*, to show the relation of the neural and alimentary canal. (Modified from Gœtte.) *n c.* Neural canal; *a l.* Alimentary canal.

This view tends to show that the up-growths known as the medullary laminæ, and the down-growths forming somatopleure and splanchnopleure represent a modification or an abridgment of the invagination process so universal among invertebrata. Among the advantages which follow upon this view of the origin of the central canal of the cord and brain is this one—it absolutely removes an objection which a few are disposed to urge against the intestinal origin of the canal in question, viz. that its epithelium is epiblastic, whereas that lining the gut is hypoblastic. In its simplest form, the hypoblast is

[1] I omit for the present any reference to the sub-notochordal rod, which is more intimately connected with the gut than the notochord.

that portion of the epiblast which after invagination lines the archenteron; following up this view of the matter, all the parts arising as modifications of the archenteron in higher and more complex animals, must naturally be regarded as hypoblastic. Thus, according to this view, the epithelium of the central canal of the nervous system from the infundibulum of the third ventricle to the extremity of the cord, that lining the neurenteric passage, the alimentary canal as far upwards as the junction of œsophagus with the pharynx, the epithelium of the trachea, bronchi, &c., the epithelium of the pleuro-peritoneal cavities, and that lining the organs derived therefrom, must be regarded as of hypoblastic origin.

His' discovery that the cells which make up the medullary folds in the embryo are not, as is usually taught, metamorphosed into nerve-cells, but form the sustentaculum—the myelo-spongium—of the nervous axis is an important fact in support of the intestinal origin of the spinal cord, and has been used with considerable skill by Gaskell in this direction.

Of course it is as yet impossible to discuss this theory in connection with the many side issues it opens up, but this is certain, it will throw considerable light upon many dark problems of pathology, and especially one which has been regarded "as linked with some of the profoundest mysteries of organic life," viz. ovarian cystomata.

THE EPILEPTIC PAROXYSM.

BY JAMES OLIVER, M.D., F.R.S. (EDIN.).

THE whole molecular world, organic as well as inorganic, exists in a perpetual state of trepidation. In the former, life is the manifestation of an inherent power possessed by its molecules of adaptation to surrounding circumstances, and it is the complete failure of this capability which renders the animate at once an inanimate object. As we ascend the scale of animal life, we witness the gradual evolution of a heterogeneous and highly instable representative structure, the nervous system, a medium of communication not only between every molecule of the body, but between every ultimate tissue of the organism and its environments, and through which, by a process of action and reaction, equilibration is maintained. In the body itself and its surroundings, there are many factors for ever at work, which tend to avert molecular stability, influenced as this is by those changes affecting the elasticity or density of the structure. If the elasticity of any body be materially augmented, we shall forthwith detect a corresponding increased proneness to be influenced by and radiate molecular disturbances; whilst any increase, on the other hand, in its density must necessarily be accompanied by a diminution and limitation of this proclivity, bearing, as these two qualities—elasticity and density—do, a direct antithesis to each other. Although the causation of epilepsy, exciting as well as predisposing, is still a question wrapt in obscurity, there can exist no doubt in the scientific mind that the manifestation of the disorder is the result of some occult change which has arisen in the whole or some part of the cerebro-spinal system. In man, the structural arrangements of the nervous system are so complex, and the functions so

entangled, that a satisfactory and withal comprehensive description of the epileptic paroxysm is rendered necessarily difficult. The most typical attacks, however, are those in which the discharge beginning in the highest nerve centres descends in a more or less markedly progressive and rapid manner, and invades the whole representative structure. The mode of onset may and frequently does vary, not only in different individuals, but in the same individual at different times; we may therefore encounter every possible variation according to the part of the nerve structure primarily disturbed, the completeness or incompleteness of its molecular instability and proneness to radiation. When a fuse is applied to any part of a gunpowder tract of like elasticity and density throughout, the molecular disturbance may travel in any or every direction. It is possible, however, to so alter in parts the density of this highly instable compound as to intercept effectually the molecular wave in its passage, and thus limit its power of propagation. This simile, to my mind, reveals well the true character of a nerve discharge, and the manner in which it may spread in those with an epileptic predisposition.

Attendant Sensorial and Intellectual Disorders.—Deriving, as we do, all knowledge of our own existence, as well as that of an outside world, through the medium of our special senses, including the tactile, of which all the others are highly organised modifications, I propose to include in a common description the disturbances manifested by, and attributable to, changes arising either in the centres receiving or in those translating impressions. Here, as elsewhere, the discharge may be more or less confined; the causal instability being limited, the surrounding structure remaining unaffected by the apparently spontaneous molecular trepidation, further radiation is prevented. The whole question of sensorial and intellectual epileptic disturbance is one wrapt in obscurity, fraught with complexity, and surrounded with difficulty, yet teeming with interest, and from a medico-legal point of view worthy of our most serious consideration.

Hitherto loss of consciousness was and still is by some considered an essential sign of epilepsy; it is, however, a

variable accompaniment of the manifestations of this disorder. In many confirmed cases I have witnessed exactly the same epileptic phenomena in the same patient, at different times associated with complete or incomplete loss of consciousness, or wholly unattended by any sensorial or intellectual disturbance whatever.

When the mental change is evinced, it is not necessarily an early symptom. Many patients retain for a more or less indefinite period of time, after the initial disturbance, a full knowledge of their own bodily state and everything that happens around them. The epileptic discharge, it is true, begins usually in the highest part of the cerebro-spinal system, and descends; it may, however, originate primarily in any part of the nervous tract, and radiate more or less irregularly. In the most marked and severe epileptic attacks, loss of consciousness is invariably complete, and is ever, I believe, in such the earliest manifestation of the paroxysm. The discharge under such circumstances is as a rule so rapidly transmitted, that the abolition of consciousness and the evolution of the motor and other allied disturbances are apparently simultaneous. The period during which there is this total interference with the reception and translation of impressions appears to, and is spoken of by, the patient as "a blank." The individual in a state of epileptic unconsciousness may or may not retain his equilibrium; he may, without any warning whatever, fall suddenly on the ground in a helpless and passive state; or he may maintain his posture, and evince no further outward manifestation other than a somewhat strange and vacant, yet withal characteristic stare and fixity of the eyes. In yet another set of cases we witness the evolution of thought, more or less coherent and intelligible, and the performances of actions more or less complete and difficult of attainment, with a total suppression of consciousness. With the initiation of unconsciousness, the individual frequently falls, sustaining at times severe bodily injuries, even fractures and dislocations. Here, doubtless, the onset is so sudden and shock-like in character, that there results a complete inhibition of the motor centres and consequent cessation of the ever-existing tone with total muscular relaxation and loss of

equilibration. In the individual who retains his position, the discharge may travel and affect the whole representative structure of the special senses, or be confined to one or more of these special sense-centres. As a rule, the disturbance in such occurs in the centre appreciating and translating impressions, and not in that receiving them. It may, however, occur in one or both, and we may encounter every possible variety. There may be complete and very transient loss of eyesight, with or without accompanying auditory change—the patient retaining the power of hearing, but being at the same time wholly incapable of comprehending and defining the impressions conveyed to the auditory centre. The auditory tract may remain undisturbed, the individual comprehending and retaining what may be said, and yet there co-exist with complete cessation of the functional activity of the visual centre a total inability to express in spoken words thoughts resulting from these auditory impressions. Mental phenomena may, however, exist and be revealed, whilst consciousness itself is in abeyance, the individual under such circumstances being a wholly irresponsible agent. Many of the actions performed during this state appear more or less automatic in character; often they are most complex, and may be exhibited apart from any evident or necessary motive.

During a state of epileptic unconsciousness I have frequently witnessed the patient run forward with much impetus, as though impelled by some irresistible power. In one case, the individual coming in contact with a bed which happened to impede the course, was thrown violently on the floor, performing a somersault, and landing on the other side of the bed. This patient sustained a severe bruise of the left hip by the fall, and continuing unconscious for several seconds afterwards, would have succeeded in recovering the erect position and rushing on again, had I not prevented her by physical force. These movements may by some be considered as post-epileptic or even hysteroid in character; that, however, they were an accompaniment and not a sequence of the epileptic state, I have no misgivings. Many patients display an awkward propensity for undressing, and it is often difficult to prevent them from throwing off every vestige of dress on the

body. This peculiarity may be a concomitant of the minor attack with mere loss of consciousness, or a truly post-epileptic phenomenon exhibited after a severe paroxysm. In one case which came under my notice—that of a female—the patient always undressed, and sometimes even got into bed when such was possible, and this whether in her own house or that of any friend or stranger. The importance of a knowledge of such facts from a medico-legal point of view cannot be overrated. The act may result from a feeling of impending illness (prior to the attack), for in the majority of our automatic movements the cell and fibre arrangement is so perfect, that the least stimulus may serve to call into action, and evoke unconsciously, with machine-like regularity, the associated train of events. The act of undressing has doubtless, through habituation, become more or less automatic. In some cases, at least, it appears to be totally unconnected with any feeling of indisposition, for one lad I interrogated whilst so undressing as to why he did so, replied, "I am going to work;" and this patient, having already thrown off his coat and waistcoat, had unlaced and taken off his boots, and was about to take off his trousers, when he recovered consciousness and re-dressed, retaining no knowledge whatever of what had transpired. Another patient, the subject of severe as well as minor epileptic attacks, had during the occurrence of a minor attack started to prepare the table for tea; he arranged the dishes with wonted regularity—a duty assigned to him some weeks previously—and was astonished, on regaining consciousness, when it was told him what he had done. Some years ago I remember a female patient telling me that, in her slight fits, she habitually—irrespective of the time of day—placed some tea in the tea-pot with hot water, and placed it by the fire, but never did she at any time remember anything of the act, nor any reason for so doing.

Our feelings, thoughts, and actions, are usually conscious; that, however, they are not invariably so, appears more than problematical. To say the individual feels without knowing he feels, is somewhat paradoxical; it cannot, however, be more so than to say the individual thinks and acts without the knowledge of thought or action, tenets which we must admit.

Although the whole structural arrangement necessary for the reception and conduction of impressions from the periphery to the higher centre is intact and functionally active, yet because the impressions so received fail to be appreciated by the organism, we say the feeling does not exist. It is not the feeling which does not exist, but the consciousness of the existence of the feeling which is non-existent.

The Cry.—The epileptic cry is, comparatively speaking, an infrequent accompaniment, but an early manifestation of the paroxysm. It is uttered in the aggregate—so far as I have been able to judge—only in about 5 per cent. of the fits. In some cases it is never noted, whilst in others it is an occasional but never a constant association. It appears to be a mere casual motor coincidence, dependent upon a state of more or less complete inflation of the lungs at the time of the initial nerve disturbance. The cry, when emitted, is very characteristic but difficult of imitation, and is a wholly unconscious phenomenon, as the patient never at any time retains any knowledge of its utterance. Sounds more or less articulate in character, and associated with the consciousness of a developing aura and consequent fit, are commonly uttered, and are most probably the result of fear or, in some few cases, apparently of pain.

The epileptic cry is an invariable manifestation of a general seizure, sudden and severe, the molecular change affecting the whole representative nerve-structure in an incalculable period of time. The muscles of the chest and vocal apparatus act in concert through habituation, the resulting automatic centre being located in the highest part of the motor nerve tract, and represented on the surface of one or other hemisphere, according to individual peculiarity in the neighbourhood of the centre for speech.

The epileptic cry, as far as my observation goes leads me to believe, is especially noted in right-handed individuals, when the nerve discharge, judging from external manifestations, originates in the left hemisphere rather than when, in the same individual, the molecular disturbance primarily affects the right cerebral lobe. In left-handed patients, again, I have invariably noted its association with an initial molecular discharge in the right hemisphere. The cry is produced by

an associated action of the muscles of the chest and vocal apparatus, whereby the air expelled from the lungs is forced through the narrow glottis. It is a true part of the fit, and may be evinced by patients equally, whether asleep or awake, at the time of onset of the attack.

Motor Disturbances in the Limbs.—In the most characteristic epileptic attacks the motor spasm, so far as the limbs are concerned, appears at first in one or other upper extremity, as the case may be, the initial manifestation being always in that toward which the head is primarily deviated. It then spreads in a somewhat regular manner to the lower extremity of the same side, to the lower extremity of the opposite side, and finally attacks the remaining upper limb. The march of retrogression is, as a rule, in the inverse order of progression, the spasm disappearing first in the group of muscles last affected, and continuing longest in those primarily disturbed. It is but seldom, however, that we witness this uniformity in progression and retrogression, the nerve structure being so complex and so irregularly liable to change, as to allow of almost every conceivable variation.

The convulsion itself is usually divided into two stages, the tonic or initial, and the clonic or sequential. Here again we rarely find the regular association, and may often detect the simultaneous existence of these two muscle states and every possible intervening gradation in the different limbs.

Experimentally, if a stimulus of regular and never-varying quantity traverse the nerve tract of a muscle, no change in its structural state, as evidenced by contraction, will result. In order that a muscle may be thrown into contraction, the force or quantity of the stimulus must vary, and according to the rapidity with which the waves of molecular motion follow each other, so do we develop a tonic or clonic spasm more or less distinct. The tonic state results if the waves of excitation are uniform, and follow each other with such rapidity that ere the influence of each preceding impulse has finally disappeared, a new wave of motion has already been transmitted. Clonic convulsion, on the other hand, is the outward manifestation of impulses, or groups of impulses, transmitted to the muscle, but separated by more or less distinct intervals, which allow of the

muscle returning after each excitation to a state of comparative inactivity.

Initial tonic convulsion, eventually replaced by clonic, is commonly observed in pure functional or so-called idiopathic epilepsy; whilst initial clonic spasm, on the other hand, is a common feature of epileptiform disorders dependent, as a rule, on some coarse cortical lesion of the cerebrum. Clonic convulsion, when a sequel of tonic spasm, apparently depends for its development upon a somewhat exhausted state of the still highly instable nerve-cells. Its frequent, though not invariable co-existence, however, with the venous turgescence resulting from interference with the respiration, renders it highly probable that the impregnation of the blood with carbonic acid may aid in determining clonic spasm through increasing the density of the disordered nerve-tissue. Tremor, more or less limited in extent, is frequently noted as a motor manifestation of the epileptic paroxysm. During the fit and under certain circumstances I have been able to develop this trepidation, especially in the lower limbs, by merely straightening the leg already slightly bent by a feeble yet maintained tonic contraction. I have also witnessed the spontaneous development of this variety of spasm or clonus, when the leg slightly bent at the onset of the paroxysm has become straightened and somewhat stiff during the fit itself. Whilst the character of the convulsion may vary, so may the extent and manner in which the muscles are involved; and this in the majority of cases without any very apparent cause. In a few cases with distinct cerebral or spinal lesion I have noted a definite alteration in the character and evolution of the motor phenomena in idiopathic epilepsy. Some years ago I saw a girl, epileptic from childhood, in whom the convulsive seizure had always been general, affecting all four limbs. At the age of ten this patient during a fit suddenly developed a complete hemiplegia (left), the result apparently of a hæmorrhage into the right cerebral lobe. In all attacks hereafter the convulsive phenomena were confined to the right half of the body. The girl is now fourteen years of age, and although she has acquired a fair amount of volitional power over the left arm and leg, the convulsion never spreads to this side.

Although prior to the occurrence of the hæmorrhage into the right hemisphere, the left arm and leg had always evinced motor disturbance, they now remain perfectly quiescent throughout the fit. Here the cerebral lesion is the undoubted cause of alteration in the paroxysm; its mode of operation, however, is at present difficult to determine. In another case I witnessed the rare association of epilepsy with paraplegia, the result of pressure from Potts' disease in the lower dorsal region of the cord. It is more than likely that in this case the epilepsy was truly idiopathic and cerebral, and wholly independent of any cord lesion. The curvature had existed from childhood, and gave rise to no material inconvenience until the age of forty-five, when complete paraplegia resulted. He regained the wonted power in the legs in the course of twelve months, and it was not until one year later that he was the subject of his first epileptic fit. The attacks thereafter recurred more or less regularly every three months, always in the night and during sleep. At the age of fifty-two, seven years after the first attack of paraplegia, he came under my notice, having again lost completely the power in both lower limbs. Gradually he began to amend, and on the day previous to an epileptic seizure I had noted that he was able to move feebly and very imperfectly the left leg, foot and toes; on the right he had no power whatever. During the fit, which was typically epileptic, the head was drawn from side to side alternately; both upper limbs were at first the seat of tonic and afterwards of clonic spasm. The left leg, at first straight and somewhat rigid, manifested during the existence of the clonic spasm in the upper extremities a general tremor, whilst the right leg throughout evinced no change. Later on, having recovered almost completely power in both legs, all four limbs alike were the seat of convulsive disturbances during the epileptic fit. It is more than probable that, in this case, the pressure on the lower dorsal part of the cord determined the freedom from motor disturbance of the right leg, and the trepidation instead of clonic spasm of the left.

Epilepsy, purely idiopathic in character, is often found co-existent with infantile or adult hemiplegia. The association may be a mere casual coincidence. In many such cases

the motor manifestation is confined to the hemiplegic side, and the fact that there is frequently no intellectual derangement whatever is indicative of an epileptiform rather than an epileptic disorder, and points to some coarse cortical lesion as the cause of the chronic motor spasm. The length of time which may elapse between the occurrence of the hemiplegia and the epileptic or epileptiform manifestations may vary. In one case which came under my notice twelve years had intervened, and here the convulsions were invariably general, and not limited or confined to the hemiplegic side. The whole question is one wrapt in obscurity, for it is quite possible that, apart from the hemiplegia, this lad might have been the subject of idiopathic epilepsy. The position of the limbs during the paroxysm may vary in the same as well as in different attacks. One or more of the extremities may be unaffected, and occasionally, although rarely, the motor evolution may be crossed, the spasm being located in the upper extremity on one side and in the lower on the other. When three limbs are invaded, it is more commonly noted in one upper and the two lower than in the two upper and one lower. Sometimes one or more limbs may be flexed and one or more may be straight. In one case the upper limbs were invariably raised above the head during the attack, whilst in another they performed a to-and-fro wing-like motion. If the patient during an approaching paroxysm hold anything in the hand, the object is tightly grasped whilst the tonic state exists, but is invariably dropped with the onset of the clonic convulsion.

Flexion is a common posture assumed, not only by the upper but also by the lower limbs. In the upper, while flexion predominates the arm is usually adducted and drawn across the front of the body, the elbow and wrist are more or less markedly flexed, whilst the thumb is bent in on the palm of the hand and the other fingers, too. When the lower limb is similarly involved, the thigh is bent on the abdomen and the leg on the thigh, whilst there is an associated extreme dorsal flexion of the foot. With strong flexion in the limbs the head is often bent forward on the chest, and so powerful and sudden is this initial force, that I have seen a patient thrown out of

bed with violence on the floor. In some rare instances I have witnessed a dominating extension of the lower limb, associated with well-marked plantar flexion of the foot so extreme as to make the foot and leg almost straight. Occasionally under such circumstances there is noted a slight disposition to arching of the back.

Micturition.—During the fit there is frequently involuntary passage of urine. This by some authorities is considered a pathognomonic sign of epilepsy; as, however, I have occasionally witnessed the association of this phenomenon with convulsive seizures, typically hysteroid in character, it cannot be viewed as an infallible symptom. Although urination is often a concomitant of the severe epileptic paroxysm with total loss of consciousness, yet the act is one which may be manifested apart from the combination, either with intellectual perturbation alone or mere motor spasm without loss of senses. In one case which recently came under my care, the patient, occasionally the subject of fully developed attacks of epilepsy, frequently experienced a mere trepidation of the nature of a general fine clonus of the right leg. In such minor attacks, and without any intellectual disturbance whatever, the bladder invariably expelled its contents, the patient retaining throughout a full knowledge of the act, but being wholly unable to prevent it. I have witnessed the bladder empty itself during simple epileptic unconsciousness unaccompanied by any motor manifestation. In a lad, the subject of minor as well as severe epileptic attacks; the minor, consisting in complete loss of consciousness and slight tremor of both upper extremities, especially the right, was invariably associated with expulsion of the vesical contents. This patient never fell during the minor attacks; but if standing, when so affected, maintained the erect position. In some cases the aura of an approaching paroxysm is the feeling of a desire to urinate, and if the act can be forthwith accomplished, the sensation passes off and no further epileptic manifestation ensues. If from surrounding circumstances the bladder cannot be emptied, the sensation may occasionally pass off, but more commonly determines a fully developed fit, in which the vesical contents are expelled. In some few

cases there is apparently a hyper-secretion of urine, the water passed immediately after the attack being paler than usual, and of unusually low specific gravity. This anomaly is probably comparable with the enormous secretion of saliva which is occasionally noted, and to which I shall presently draw attention. On three occasions I have detected a slight trace of albumen in the urine after a paroxysm, but never any trace of sugar.

The emptying of the bladder during a fit apparently depends on the state of distension of this viscus at the time of onset. In some cases it appears to result from an inhibition of the bladder sphincter. As a rule, however, it is effected by a direct contraction of the bladder muscles, aided by the superincumbent pressure exerted by those of the abdominal walls.

Erection of Penis and Emission of Semen.—In severe attacks of epilepsy I have witnessed on several occasions erection of the penis, but never at any time ejaculations of semen, nor have I been able to detect after the fit any evidence of its emission. It is possible, however, that should the circumstances favourable for the act exist, emission may result. Erection appears to result from a general involvement of the body structure, as I have never noted its presence in mild attacks.

Defæcation.—The involuntary passage of fæces is an act of very rare occurrence during the epileptic fit, as I have only once noted it in the many hundreds which I have witnessed. In this case the copious evacuation which resulted leads me to believe that the mere tonic contraction of the body muscles as a whole determined the expulsion of the contents of the rectum, apart from any special contraction of the intestinal tract or inhibition of the sphincter muscle.

Salivation.—A copious flow of saliva is a frequent accompaniment of the epileptic paroxysm, and is most commonly noted in those fits which are preceded by a distinct epigastric aura. As a rule the secretion is thin and watery, characters exhibited by that produced by excitation of the chorda tympani nerve, or associated with a distinct feeling of nausea. In several cases the saliva has literally poured out of the

mouth, its downward flow into the stomach being prevented by the existence of œsophageal spasm, to which I shall presently draw attention. If the tongue has been bitten, the saliva may be tinged with blood. In a few rare instances I have witnessed a markedly sanguinolent discoloration of the secretion, and yet have been unable to detect any evidence whatever of laceration of the tongue. Under such circumstances the blood is probably the result of a transudation from, or a direct rupture of the blood-vessels situated somewhere in the pharynx.

The secretion of saliva during the epileptic fit appears to be of a purely nervous character, and wholly independent of any direct alteration in the blood-supply of the glands. Increased blood-supply is ever a necessary concomitant of augmented functional activity, but what I mean to infer is that the alteration in the vascular state of the gland is not the initial cause of the hyper-secretion, but merely an essential association. The excitation is probably purely central or cerebral, and not truly reflex, and is apparently unconnected with motor disturbance even of the jaw. I have remarked its presence during a distinct pallor, as well as marked flushing of the face.

Abortion.—In a few rare instances I have noted perfect immunity from epileptic attacks in females during gestation; as a rule, however, this physiological state interferes in no way with the recurrence nor character of the paroxysms. Women who suffer from epilepsy are invariably most prolific, and it is somewhat astonishing that although the whole body structure appears to be more or less invaded by a severe paroxysm, the uterus, then in a state of gestation, is seldom so involved as to expel its contents. The epileptic fit itself I have never known to be the initial factor in producing abortion. The accident, however, occasionally happens, and is commonly the result of some direct injury sustained during the fall.

Vomiting.—Emesis I have never witnessed during the epileptic seizure, although I have in a few rare cases noted its occurrence before and after the fit. The symptom is ever a most dangerous one, as food is apt to pass into the glottis, and bring about a fatal result. When we remember that salivation

is a frequent concomitant of the paroxysm, it is surprising that vomiting never occurs. In order that the contents of the stomach may be expelled, the cardiac orifice must remain patent, and this probably never happens during a fit. Contraction of the gullet and cardiac end of the stomach is apparently produced before the gastric organ itself becomes invaded or affected by the superincumbent pressure of the diaphragm and abdominal walls. In a few rare instances, patients who during a meal have felt the usual epigastric aura common to them before a fit, have informed me that they have then occasionally experienced some difficulty in swallowing, a fact strongly in favour of early spasm of the gullet.

Vascular Changes.—Palpitation is often a precursor of the epileptic seizure (minor as well as severe), and in some cases it even constitutes the distinct aura or warning of an approaching paroxysm. During many fits in the male and female alike I have detected a marked increase in the frequency of the pulse, reaching, though rarely, as high as 120 or 140 per minute.

After the cessation of the attack, it gradually lessens in frequency and returns to its normal rate. Augmentation of the pulse is more commonly observed than diminution; in a few rare instances, however, I have found a pulse averaging as a rule 76 per minute reduced during the actual fit itself to 48. In many cases I have remarked no apparent or rather appreciable change, whilst on one occasion only did I note intermission in a pulse which at other times was perfectly regular.

In some cases there is either a marked pallor or a distinct flushing of the face, indicative respectively of contraction and dilation of the blood-vessels; occasionally, however, during the same attack, I have observed an alternation of these two vascular states. During a severe paroxysm the face, irrespective of its initial colour, is invariably livid, because of the interference from muscle spasm with the pulmonary circulation. The cyanosis gradually lessens, and the face re-assumes its normal colour as the clonic spasm becomes more completely interrupted.

During severe fits, hæmorrhages, more or less extensive, may

result. In the skin, these usually partake of the character of petechiæ, and are most commonly detected on the neck, the eyelids, and external ears, especially the lobes. On one occasion I witnessed a distinct and, comparatively speaking, profuse hæmorrhage from both internal ears. Not infrequently I have noted extensive sub-conjunctival hæmorrhages in one or both eyes, which underwent during the process of absorption the usual changes incident to such. Occasionally, as I have already remarked, rupture of blood-vessels occurs in the pharynx or œsophagus.

Respiration.—In minor epileptic attacks, with mere loss of consciousness and slight flushing of the face, I have frequently observed a marked acceleration of the respiration. As a rule, however, in the typical attacks, there is interference with the respiratory movements, and signs more or less distinct of asphyxia result from a fixation of the respiratory muscles and spasm of the glottis. In many cases, the asphyxiated state becomes so marked, that death seems inevitable.

The state of the Reflexes.—During the epileptic fit there is total abolition of all reflexes, the pupil fails to respond in the usual manner when exposed to a strong light; the conjunctiva, when touched, fails to elicit a reflex contraction of the orbicularis; and, when favourably tested, the plantar surface of the foot when tickled, fails to produce its wonted contraction of muscles.

Finger Snapping.—Finger snapping is an odd gesture, yet I have once witnessed its manifestation during the epileptic unconsciousness. It is supposed to be a sign indicative of contempt; whether there had been any such idea in the mind of my patient prior to the exhibition of the above peculiar phenomena I failed to elicit.

The state of the Pupils.—During the actual fit itself the pupils are, as a rule, widely dilated; on two separate occasions only have I witnessed marked contraction, a condition which must be considered as very rare. In a lad aged eight, the subject of minor as well as severe and general epileptic attacks, I had the opportunity of witnessing several paroxysms. During and after the minor fits in this case, it was difficult, nay utterly impossible, to elicit with certainty whether uncon-

sciousness was complete or but partial. I believe there was mere intellectual disturbance, the result of limitation of the epileptic discharge to the left cerebral lobe, deranging the higher centres of this hemisphere alone. In the minor attacks he was totally unable to speak, yet appeared not wholly unconscious, but seemed to comprehend, although somewhat imperfectly, what was said to him. The sole muscle manifestation of the paroxysm was a twitching of the orbicularis of the right eye, associated with a dilation of the pupil of the same side, whilst that of the left eye was unaffected throughout the attack. In this case the dilated pupil was on the side towards which the head would probably have been primarily deviated, and away from that hemisphere, the seat of initial discharge. Associated activity of the two hemispheres of the brain is the rule, yet it is possible, and the above case somewhat favours the acceptance of the theory, that dual and independent action is probable. In this lad, the dilation of the right pupil in the minor attacks may be considered as comparable with the dilation which nominally results when an individual is directed to look into space. The pupillary change in this case suggested to me the possibility of my being able to derive some valuable information from the pupil states—so constantly associated with epilepsy, and to which I shall presently refer—in determining approximately the seat of initial cerebral disturbance.

Dilation of the pupils, as is well known, occurs during violent muscular effort; it is, however, a noteworthy fact, that this state of pupil is a concomitant of the minor attack as well as the severe epileptic paroxysm. Pupillary dilation was at one time considered to be dependent on a contraction of the blood-vessels of the iris. Now were the old theory admissible, that contraction of the blood-vessels of the brain, as manifested by a pallor of the face, is the initial change in the epileptic fit, dilation of the pupils, as a result of contraction of the blood-vessels of the iris, might be feasibly maintained. During ordinary blushing the vessels of the retina and the brain have been seen to dilate; it is more than problematical, therefore, that the vessels of the iris are similarly affected. If, however, the pupillary state were the result of alteration in the

calibre of the blood-vessels circulating in the iris, we should expect to find dilation of the pupil associated with epileptic pallor, and diminution in size co-existent with the initial flushing. Although contraction of the pupil has been noted during the fit, the condition is extremely rare, and marked dilation is the rule, whether there be manifested an initial pallor or flushing of the face. The pupil state in epilepsy appears to me to be wholly independent of the sympathetic influence, also totally unconnected with any cord change whatever—and is most probably of a purely cerebral origin.

In many cases of epilepsy I have frequently remarked, during the period of immunity from attacks, the existence of a permanent inequality of the pupils. I have also, it is true, occasionally observed an inequality of the pupils in individuals of apparently good health; but the common association of this phenomenon with epilepsy makes it somewhat problematical that it may be a direct result of the proneness to this chronic functional disorder. In some cases, I have noted that the pupils, apart from any attack, are ever widely dilated, and either do not react at all to light or but feebly, the nature of the contraction at the same time being wavy and uncertain. The pupil under such circumstances displays a marked proneness to dilate again, the contraction produced being most fleeting and transitory, which otherwise in good health would continue so long as the exciting cause lasted. When the pupils are unequal, the least effort at accommodation may determine equality, the inequality only reappearing with the state of comparative rest. In testing the reaction to light, I have frequently remarked that whilst the pupils may in conjunction react well to this excitation, separately the one more dilated will be found to contract with less certainty and less markedly than the smaller, and at the same time manifests a great tendency to return forthwith to its pre-existing state of dilation. The iris itself is a highly organized structure, and as the epileptic may be considered as epileptic even to the finger tips, it is quite possible that this protoplasmic veil in the eye participates in the general structural instability. So far as my own observation goes, a careful scrutiny of those cases with inequality of pupils reveals no constant information

regarding the seat of initial cerebral disturbance as manifested by the primary deviation of the eyes and head.

The dilation of the pupil during the actual fit results probably from a withdrawal of the usual nerve-toning influence, whilst the permanent, wavy and uncertain state of the pupils, so constantly observed in epileptics, is the result of some occult tissue-change in the iris itself.

The Eyelids.—During the fit the eyelids are usually open; on several occasions, however, I have observed a closure of one eye not only during the state of tonic contraction, but also during the existence of clonic spasm. The eye closed is invariably that towards which the eyes and head are primarily deviated, the other eye remaining open throughout the attack. In a few cases I have seen both eyes firmly closed by spasm. During violent exercise, screaming, sneezing, and so forth, it has been suggested that closure of the eye produced by contraction of the muscles is for the purpose of protection—a wise provision of nature to prevent rupture of the over-distended blood-vessels. Rupture of the blood-vessels of the eye, deep or superficial, is, however, an accident of so unfrequent occurrence during the fit, when the eyelids are as a rule open, that I question their alleged protective efficiency when closed. Closure of the eyelids is effected more especially by a descent of the upper lid, and I am inclined to think that during the epileptic fit closure is prevented by a dominating and early contraction of the levator palpebræ.

INHIBITION.

BY CHARLES MERCIER, M.B.

Of all the Functions of the Nervous System none is so mysterious in its nature or so obscure in its working as that of inhibition. The difficulty of comprehending the method of its action appears to me to have been greatly enhanced by the circumstance, that the most striking and palpable instance of the process, and the one that first arrested attention, is the case of the inhibition of the action of the heart, which is probably an unusual and exceptional form of inhibition. Thus it has happened that the classical example of the process is one that cannot readily be compared with others, nor can we readily argue from this to other instances, and so we are deprived of a powerful leverage.

In order to gain a clear concept of the true nature of inhibition we must go down to the bed-rock of the fundamental properties and powers of the nervous system, and regard it as an apparatus for the storage and expenditure of force. If we regard each cell as a reservoir in which force is stored, and each fibre as a channel by which force is carried, we start from a position which no one will nowadays impugn, and which affords us a secure foundation on which to build an hypothesis of the nature of inhibition.

The first course to be laid down on this foundation is the fact, also undoubted, that the expenditure, or release, or emission, or escape, of force from the nerve cells, never occurs continuously, but always in intermittent, interrupted outbursts. Whether we regard the process in the aggregate or in detail, this fact is apparent. Every part of the body has its periods of activity and its periods of repose. That is to say, to every

part of the body force is distributed by the nervous system, at some times in abundance, and at other times in driblets. Every muscle has its intervals of contraction and its intervals of rest. That is to say, to every muscle force is occasionally distributed in large quantity, and occasionally in small. Furthermore, if we look at the intimate composition of each muscular contraction we find that it is similarly constituted. For each tetanic muscular contraction is made up of a large number of simple contractions following each other very closely; that is to say, the current of force which causes each muscle to contract is not a continuous current, but an interrupted current—a succession of separate shocks. So that we arrive at this conclusion:—that the force which is expended by the nerve cells is released, not continuously, but in an interrupted current—in a succession of waves, or rather, more strictly speaking, of bubbles, for there is every reason to suppose that each delivery is entirely separated from its neighbours.

This interrupted delivery of force from the nerve-cells is very remarkable, very significant; and if I might venture to express the opinion, has hardly yet received sufficient attention. Its great significance consists in this, that while the *delivery* of force is interrupted, the *storage* or accumulation is undoubtedly continuous. For while the delivery of force is due to the fall of atoms into simpler combinations, the storage of force is effected by the rebuilding of atoms into combinations that are more complex and unstable. This building up of complex molecules is an instance, and a part, of the function of nutrition, a function which, while it varies in activity from time to time, never, as far as we know, ceases altogether. Hence it appears that while force is continuously being stored in every cell, yet it is delivered not continuously but only from time to time; and when delivered, the delivery is not a continuous stream, but an interrupted bubbling over. The question arises, How is this interrupted delivery to be accounted for?

If we look round the world of physical phenomena, we shall find that there are several other instances of a continuous accumulation, both of matter and of force, being broken up

into an interrupted delivery. A continuous stream of gas poured into a liquid escapes as an interrupted series of separate bubbles. Similarly a continuous stream of water falling through the air is broken into an interrupted series of separate drops. The continuous pressure of a stream against a water plant causes, not a steady bending, but a waving to and fro of the leaves. The continuous pressure of the steam on the lid of a kettle causes the lid to rattle up and down with an interrupted vibratory movement. So, too, the continuous tension of electricity on the prime conductor of an electric machine escapes in an interrupted series of separate sparks.

In all these cases, and in every other case in which a continuous accumulation is broken into an interrupted delivery, we find always the same result produced by the same conditions. And the conditions are the opposition of a continuously accumulating force to a resistance that is constant. The continuous accumulation of the gas at the end of the pipe is opposed to the constant pressure of the water, and we get a series of bubbles. The continuous increase of the velocity of the falling water produces a continuously increasing pressure of the air against it, which is opposed by the constant resistance of the weight of the water. The tension of the leaf stalk, increasing continuously as it is bent, is opposed to the constant resistance of the stream of water, and we get a series of rebounds. The continuous accumulation of the pressure of steam in the kettle is opposed to the constant resistance of the weight of the lid, and we get a series of puffs of steam escaping. The continuous accumulation of electric tension on the prime conductor is opposed to the constant resistance of the air, and we get a series of sparks.

It appears therefore reasonable to advance, as a working hypothesis, that the intermittent delivery of force from the nerve-cells in which the force continuously accumulates, is owing to the opposition of a constant resistance, as in the many other cases in which a similar effect is produced. The state of things supposed by this hypothesis will involve certain other consequences besides the intermittent delivery of force, and if we find that the facts are in accordance with the necessary consequences of such an arrangement, we may

proceed with some confidence still further to utilise and apply the hypothesis.

If we take the rattling kettle off the fire and put it on the hob, so that we bring the contents from a boil to a mere simmer, we find that we stop the rattling at once, but that every now and then the lid gives a jump and allows a puff of steam to escape. By taking it off the fire we have caused it to "go off the boil," that is to say, we have diminished the rapidity of the production of steam, we have diminished the rapidity with which the tension accumulates, and, the resistance still remaining constant, we have diminished the rapidity with which the outbursts of steam succeed one another. Similarly, if we turn the disc of the electric machine more slowly, the sparks remain of the same intensity, but they succeed one another at longer intervals. With a slower accumulation of tension, a longer time is consumed in arriving at a sufficient strength to overcome the resistance, and consequently the outbursts are farther between. But since the resistance remains constant, the strength that has to be arrived at to overcome it remains constant, and each outburst is of precisely the same intensity as the others.

Is there anything analogous to this state of things in the operation of the nervous system? Do we ever find that the evidence of the outbursts of force from the nerve cells tends to show that these outbursts, while retaining their usual intensity, occur more slowly? I think such evidence does occur. The effect of each separate outburst of force from the nerve cells is to produce a single simple contraction of the muscle. Normally these simple contractions follow one another so fast that they are all fused together into a continuous tetanic contraction; but if they follow one another more slowly they will not be so completely fused together. The vibration which is so fine and rapid as to be imperceptible, as it gets slower will get coarser, and will become perceptible. So that instead of a continuous muscular action there will be a vibratory or tremulous muscular action. In short, there will be *tremor*. If the hypothesis be true that the nervous discharge occurs by the continuous accumulation of a tension against a constant resistance, there ought therefore to be tremor whenever the rate of accumulation

of tension is diminished. And what are the circumstances under which tremor occurs? It occurs in fatigue; that is to say, after long continued discharge has exhausted the nerve cells of their usual surplus of tension, and after great demands have been made not only on the amount of energy stored up in the cells, but on the process of storage, which has had to keep pace with an unusual demand. The process of storage being continuous, while the process of expenditure is intermittent, the former process is of course and of necessity slower than the latter; hence if expenditure continues, it must at length exhaust all the energy actually in store, and further demands will have to be supplied from hand to mouth, as it were, by the simultaneous process of storage, which as we have seen is normally slower than expenditure. So that when nerve cells have been discharging for some time, the rate of accumulation of tension in them must diminish; and it is when they have been discharging for some time that tremor occurs. Tremor also occurs in exhausting illness, when the general process of nutrition all over the body is weakened, and when the whole bodily condition indicates a lack of intensity of the nervous discharge. In paralysis agitans the whole nervous system does its work badly; there is great and general loss of nervous vigour; that is, there is evidence of slackened accumulation of tension in the nerve-cells. And in paralysis agitans there is tremor. In drunkenness there is tremor, and tremor is worst at the time that every symptom points to the lowest ebb of nervous energy—in the early part of the day.

So far, observed facts corroborate the hypothesis, that the nervous discharge occurs on the breaking down of a constant resistance. We have supposed the resistance to remain constant while the rate of accumulation of tension varies. But suppose the resistance varies in magnitude, what will be the result? Returning to our illustration of the electrical machine, we find that when resistance is increased by increasing the distance that the spark has to travel, two results occur. The tension must reach a higher point before it can overcome this increased resistance; and if the handle be turned at the same rate, then in order to reach a higher point, the tension must have a longer time to accumulate. So that one effect of increasing the resist-

ance will be to increase the intervals at which the shocks are given out—to render slower the succession of shocks. Also, since the tension must overcome the resistance before the discharge can take place, and since the resistance is increased, the tension at the moment of discharge must be increased. So that as the resistance is increased the shocks of discharge become stronger but wider apart. As we increase the resistance of the air by increasing the distance that the spark has to travel, we increase the magnitude of the sparks and the loudness of their report, but we render their succession slower. As we increase the resistance to the escape of steam from the kettle by putting a weight on the lid, we cause the lid to make bigger jumps, but the jumps follow one another more closely than before.

In the case of the nervous system we cannot alter the resistance at will, and indeed our only means of judging of increased resistance would be by noticing a concomitant variation in the strength and the rapidity of the simple contractions; so that we are in danger of arguing in a circle. But this we can do: we can see if there is any condition in which the magnitude of the simple contractions bears, while varying, a direct proportion to the varying magnitude of the intervals between them; and if such a concomitant variation is found, we may justly argue, first, that it completely harmonises with the hypothesis with which we started, and second that it is difficult to conceive any other disposition of forces that would so completely and satisfactorily and simply account for all the phenomena.

The concomitant variation in the magnitude and rapidity of simple contractions is found in the course of almost every epileptic fit. In an epileptic fit, beginning in the face, for instance, the opening spasm is "tonic" in character; that is to say, the movement of the part affected is even and regular, like an ordinary natural movement. The eyes turn to one side, or the corner of the mouth is raised, with a movement so gentle and natural, that but for what follows, we should not know that it was part of an epileptic fit. The next thing is that the eyelids or cheek, as the case may be, begin to quiver with a fine vibration. The simple contractions are beginning to show separately, and the spasm is manifestly stronger. As the fit progresses, the fine vibration becomes coarser and

coarser, and at the same time the spasm becomes stronger and stronger, producing greater and greater contortions. By the time the limbs have become involved, the spasm has become markedly chronic in character, and of great violence. Now, as the fit passes off, the several shocks come more and more slowly, but assume a greater and greater intensity, until at length a condition of complete and universal relaxation and flabbiness separates from one another the shocks, which coming now at the rate of only one or two in a second, are of tremendous violence, and seem as if they would break the very bones. At last one terrific universal spasm, coming and going with the electric suddenness that characterises a simple contraction, ends the fit.

So far as the concomitant variation in the strength and rapidity of the shocks can support the hypothesis of nervous action here advanced, the phenomena of such a fit as that described, which is a very common one, do corroborate it completely. If explained by the hypothesis, the phenomena suppose, of course, that the resistance to the discharge gradually increases from the beginning to the end of the fit; and without speculating as to how this increase takes place, it appears extremely probable that it actually occurs. And if it do occur, then we draw this important conclusion: that in the balance of tension and resistance which we suppose to exist in the grey matter, not only does the rate of accumulation of tension vary, but the amount of resistance also varies from time to time. That the accumulation of tension varies both in health and in disease has already been demonstrated; and although the only variation of resistance that we have been able to identify has been a morbid variation, yet, on the general principle that morbid variations are but exaggerations of normal variations, there can be no reasonable doubt that, if the hypothesis is true at all, it is true that both the rate of accumulation of tension and the amount of resistance to its discharge are subject to variation within the limits of health.

Here, then, we appear to have got an inkling of the way in which inhibition may possibly be produced. If all nerve-cells are furnished ready to hand with a resistance to their discharge which is variable within the normal, then it is

conceivable and possible that inhibition may be due to an increase of this resistance up to and beyond the limit of the tension attainable by the cells. There is just a trifle of evidence in favour of this view. Supposing inhibition to be effected in this manner, and supposing in any given case a great increase of resistance to prevent a given discharge; and supposing further a sudden increase of tension up to and beyond the increased resistance; the result will be an outbreak of great violence—of greater violence than if no inhibition had been exercised—for it will take place in the teeth of a greater resistance. I think that experience, to some slight extent, enforces this view. If we have tried hard to stifle a laugh or a yawn, and it at length breaks out in spite of the inhibition, it is more violent than if we had at first given way to it; and the same is surely true of an outbreak of temper.

Leaving now the purely physical aspect of the subject, let us turn to the physiological side, and see what evidence can be obtained of the way in which inhibition occurs.

Supposing the nerve cells to be grouped into clusters, each of which, with its fibrous prolongations and intervening grey matter, exercises a specific influence over a specific part of the body, and is called a nerve-centre, then the most elementary and obvious fact, and at the same time the most significant fact, in connection with the action of nerve centres is that they are not continuously and equably active, but alternate between activity and rest; and if active, their activity may vary much in volume and intensity. The initiation of activity of a nerve-centre is easily accounted for. Its equilibrium is upset, its discharge is started, its activity set going, by the impact upon its elements of a current of force arriving from without,—arriving either directly from the external world through the organs of sense, or indirectly, through the intermediation of other nerve-centres. But how are we to account for the arrest of the activity? If it is suggested that the centre subsides spontaneously by virtue of its inherent internal organisation to a state of rest whenever the stimulus to activity ceases, the answer is that, while there is no evidence in favour of such a structure, there is this powerful argument against it, that it is opposed to the most fundamental laws of physics. The first

law of motion is surely as true of nerve-centres as of planets—that a body at rest will continue at rest, *and if in motion will continue to move, ... unless acted on by some extraneous force.* The natural inherent tendency of a nerve-centre that has begun to discharge is to go on discharging until it is utterly exhausted. There is no mechanism discovered, I will venture to say no conceivable mechanism, internal to the centre itself, which will cause the centre to spontaneously cease discharging, and yet leave it ready to discharge again on slight provocation. Once set going, it must, unless arrested by some external agency, continue to discharge until emptied of its store of force. But this never occurs normally in the healthy organism. Practically we never become so exhausted by performing any given action that it is impossible to perform it once more. A single touch of the eye-ball starts an action of a nerve-centre which actuates a single blink of the lids, and then the action ceases, and the lids are again opened. If the touch is intense, or is repeated, there may be several blinks in response to the irritation; but when the irritation at length ceases, the movement ceases also. Why does the movement cease on the suspension of the irritation? If there is nothing, as I submit there can be nothing, in the constitution of the centre itself to cause the arrest of its action when once it has been set going by the impact of an extraneous force, then the arresting action, like the initiating action, must come from outside. It must be imposed from without. Hence we gather that *every nerve-centre is normally subject to an inhibitory influence imposed upon it from without.*

A moment's consideration will show that this inhibitory influence is not a momentary and occasional phenomenon applied only from time to time when the cessation of activity is required. It is now an universally accepted doctrine that the whole of the nervous system is at all times the seat of a continuous circulation of currents of force of greater or less intensity. Upon every centre are continually breaking waves of force of varying intensity, some of which, taking into consideration the various degrees of instability attained by the nerve-centres, would frequently set a centre discharging at odd moments, without reference to other movements going on

unadapted to any end then sought after by the organism. In short, we should all be continual sufferers from chorea. As we do not so suffer, we must therefore suppose that the application of the inhibitory influence upon a nerve-centre is not an occasional and intermittent incident in its history, but that it is continuous. That all nerve-centres are at all times subject to continuous inhibition or control, which steadies their action, prevents their discharge being initiated by inadequate impulses, and insures their return to a state of quiescence when the call to activity has ceased.

Lest this hypothesis of a universal and ever-present inhibition should appear too startling, let me hasten to explain that it is not new. The frequent use of the phrase "Loss of control" (indeed I have heard an eminent member of this society speak of "an outburst of loss of control") has surrendered the whole position. For control to be lost it must first be present; and if maintained upon some centres, why not upon all? The hypothesis already exists, therefore, and is already largely accepted; and, indeed, it is of such indispensable necessity to a comprehension of the physiology of the nervous system, that we may say of it, as the French philosopher said of the Deity: "If it did not exist it would be necessary to invent it."

We are now in a position to combine the conceptions gained of the physiological mode of action of the nervous system with the physical mode of action already arrived at, and to conclude that while every nerve-cell has its inherent tendency to discharge for ever opposed by a continuous resistance, so every nerve-centre has its inherent tendency to discharge for ever opposed by a continuous inhibition; and it is not making a very large demand upon our imagination to suppose a certain community of nature between actions so similar. In short, we may fairly suppose, that the inhibition is a strengthening of the resistance to the discharge of all the cells throughout the centre.

Thus we find that, while the intrinsic tendency of every nerve-cell and nerve-centre is ever to discharge, this tendency is continuously counteracted by an extrinsic influence which curbs it into quietude. Just as, in molecular physics, atoms are maintained in a condition of mobile equilibrium by the

opposing forces of attraction and repulsion; just as, in molar physics, the planets are maintained in their orbits, that is, in mobile equilibrium, by the opposition of gravitation to their own inertia; so the nerve-centres are maintained in a condition of mobile equilibrium by the opposition of the inhibition exercised upon them to their own tendency to discharge.

Very little consideration is required to show, that to have its nervous centres arranged in a condition of mobile equilibrium is vastly more advantageous to the organism than to have them disposed in any fixed arrangement. In the first place, a mobile equilibrium is far more stable, in the sense of being less easily destroyed, than any fixed disposition. The phenomena of the gyrostat are familiar illustrations. A spinning top remains upright on its point without any external support, in spite of the inclination or the movement of its supporting surface, and in spite of deflections of its axis from the perpendicular, even of considerable magnitude. The same top, if not spinning, cannot be balanced on its point for an instant, and even, if by superhuman patience and steadiness it were to be so balanced, the slightest breath of air would at once cause it to fall.

While, on the one hand, the moving equilibrium presents a maximum of stability, on the other hand, paradoxical as it may appear, it presents a maximum of plasticity; that is to say, while it is with difficulty destroyed, it is modified and altered, within limits, with the greatest ease. Consider, for instance, the example of mobile equilibrium presented by the circulation of the earth in its orbit. So delicate is its balance that the movement of another planet, hundreds of millions, thousands of millions, of miles distant, produces a sensible perturbation in the path of our earth. It is as sensible to the presence of other bodies at these inconceivable distances as a magnetic needle is to an iron bar in its neighbourhood (another combination in mobile equilibrium). Suppose now that the earth, instead of circulating freely in mobile equilibrium, were fixed to the sun by a rigid rod. If all the other planets were massed together and brought within a mile of the earth's surface they would scarcely produce any perceptible alteration in its position in space. These examples will be sufficient to

show how susceptible an arrangement in mobile equilibrium is to external influences; how readily and completely it returns to the normal after disturbance, and how difficult it is to break down. When it is remembered that the two fundamental properties of the nervous system and of nerve-centres are those of being readily disturbed from without, and of readily resuming their *status quo ante*, and when it is further borne in mind that this susceptibility to disturbance must be united with great resistance to disintegration, it will be seen how immensely advantageous it must be to the organism to have its nerve-centres in that condition of mobile equilibrium in which these antagonistic qualities are so prominent and so closely united.

Granting that every nerve-centre has its intrinsic tendency to discharge continually checked, counteracted, and controlled by an extrinsic inhibiting influence, two questions remain: What is the nature of this influence, and whence is it derived?

As to the nature of the influence, we have already considered its probable alliance with the normal resistance to the nervous discharge, but this does not carry us very far, since we are scarcely yet in a position to speculate as to the nature of this resistance. There is, however, another source from whence we may obtain a glimpse of light upon the process. This is the unquestionably undulatory character of the currents of force in the nerve fibres. Once admit, and every one does now admit, that the nerve currents are undulatory in character, and at once the whole doctrine of interference of waves is at our service to explain the phenomena of inhibition. It is more than probable that it is in this doctrine that the detailed explanation of inhibition will at length be found, and in the meantime it gives us valuable help, for just as two rays of light from the same source may be united to produce darkness, and just as two rays of sound from the same source may be united to produce silence, so we may imagine that discharges from one source may, according as they are combined, reinforce or neutralise each other.

Leaving the intimate nature of the inhibitory influence on one side, as a problem for the present insoluble, let us turn to

the other problem, and ask what evidence we have of the source from whence the influence is derived. Two hypotheses are admissible. Either there may be one or more portions of gray matter specially reserved and set apart for the purpose of producing this continuous inhibition of all motor centres, thus constituting special inhibitory regions; or the function of inhibition may be carried on by the ordinary motor centres concurrently with their more generally recognised functions.

Of the existence of a single centre, whose function is inhibition and nothing else, there is no evidence, and from the nature of the case there could not but be evidence if such a centre existed. Suppose a sudden lesion, such as a blood clot, or an experimental injection, destroyed such a centre, the effect would be that the whole control would instantly be removed from every motor centre in the gray matter, and the consequence would be such a terrific spasm of the entire body as the severest epileptic fit can give us but a faint and ineffectual idea of; the spasm would at once arrest the breathing, and speedy death would surely follow. Such a malady is not clinically known, nor have all the localising experiments of Dr. Ferrier revealed the existence of any such centre. Neither is there evidence of such a centre being destroyed by a gradually invading lesion. In that case there would be excess of movement gradually increasing to universal spasm, and ending in death either by exhaustion or by arrest of breathing as before. Something corresponding to this description does actually take place in chorea, and it is at first blush conceivable that chorea might be due to the morbid weakening of a general inhibitory centre, but such an hypothesis will not bear investigation. For the movements of chorea, although affecting all parts of the body, are manifestly not due to discharge of all motor centres. Apart from their elaborate character, there is the fact that they are essentially *a succession of different movements*, and indicate therefore a succession of discharges of different centres. Whereas the weakening of our universal inhibitory centre must produce a simultaneous discharge from all centres.

The inhibition of motor centres must therefore be actuated not from a single store, but from many different places; and

the next question is whether there are regions of nerve-tissue specially set apart for the origination of inhibition alone, or whether this function is exercised concurrently with others? It may be said at once that the balance of the evidence is overwhelmingly in favour of the latter hypothesis. Neither in experiments on animals nor in the experiments exhibited by disease and accident has there been any instance of a destruction of gray matter producing primarily an outburst of movement; and it is in the highest degree improbable if a number of centres existed, and by hypothesis the number must be large, whose functions were purely inhibitory, that no one of them should ever have been singled out for destruction by experiment, by accident, or by disease. Hence we are driven by a series of exclusions to the opinion, that the state of inhibition, which universally obtains throughout the motor centres, is maintained by centres which exercise this function concurrently with others.

In corroboration of this opinion there is a very large body of evidence from pathological observations. Experience at the bedside shows absolutely invariably and without any exception whatever, that whenever nervous tissue is destroyed the effect of that destruction is twofold. There is always a loss. The body or some part of the body is unable to do something that it could do before. The function of the destroyed part is abolished. In addition to this simple and direct effect of the lesion there is always, and without exception, another effect. There is always increased action of the body or of some part of it. These two results of simple destruction of tissue are called by Dr. Hughlings-Jackson the negative effect and the superpositive effect. It matters not to what pathological agency the destruction of tissue or of function is due; whether the nerve tissue is ploughed up by a blood clot, or smashed by a blow, or consumed by a morbid growth, or whether the function alone is abolished by chloroform or by ether, or by alcohol or other drugs, or whether the nerve region is merely boycotted by the severance of its communicating fibres. In any and every case in which nervous influence is withdrawn we find this double effect: the negative effect and the superpositive effect. If we cut a peripheral nerve we abolish nervous

influence in the parts that nerve supplies We find that the muscles supplied by the nerve are paralysed (negative effect). And we find that the reaction of these paralysed muscles to galvanic excitation is increased (superpositive effect). If we cut the cord, we get the negative effect of paralysis in all the parts below; and we get the superpositive effect of increased reflex action in the paralysed parts. Rupture of an artery in the corpus striatum gives rise to destruction of the tissue, and the sudden shock, with increase of pressure in the cranium, produces the apoplectic state. In the apoplectic state the whole of the voluntary muscular system is paralysed (negative effect); and the movements of breathing, the action of the heart, the secretion of the skin, are exaggerated (superpositive effect). When the apoplectic state passes off, there is hemiplegia and there is exaggeration of reflexes. When the highest nervous centres are placed out of action, either by destruction, as in general paralysis, or by suspension of function, as in alcoholic poisoning, there is an inability of the individual to do any action of high elaborateness and complexity (negative effect); and there is uproarious conduct (superpositive effect). On all levels of the nervous arrangements and in all classes of destroying or suspending lesions the rule holds absolute.

The abolition of a function is of course sufficiently accounted for by the abolition or destruction of the organ that affected that function. The negative effect of a lesion is easily accounted for. But how are we to account for an *increase* of action consequent on a *destruction* of tissue? It is manifest that no amount of destruction of tissue can of itself primarily produce increased action. In other words, the superpositive effect cannot be due to increase of action of the part destroyed. That part, so far as function is concerned, has ceased to exist. The superpositive effect must be due to increased action of some other part. Why does this other part overact when the first part is destroyed? Surely there can be but one answer; because the part destroyed exercised a controlling, inhibiting, moderating action upon the part that overacts; and the sudden outbreak of overaction is due to the sudden removal of this permanent controlling influence.

There is another circumstance with regard to the double

effect of a destroying lesion which is of the utmost significance. There is always a constant relation between the negative effect and the superpositive effect. The action that is increased is always of a simpler, more fundamental, less elaborate, more general character than the action that is lost. Motion is lost, galvanic reaction is increased. Movement is lost, reflex action is increased. The voluntary muscles are paralysed, the involuntary muscles overact. The most elaborate forms of conduct are lost, the simpler forms are exaggerated. So that it appears that every nervous centre is subject to the control of a more complex and elaborate centre. In other words, the nervous centres are arranged in a hierarchy like the ranks of an army, every grade not only supplying an initiative to, but also exercising a control upon all grades below; and every grade being subject to the control and stirred by the impulse of all grades above.

There is, I submit, nothing anomalous or paradoxical in the supposition, that the same centre affords both the impulses which start and accelerate, and those which retard and arrest the action of inferior centres; for we have a daily illustration of a similar condition of things in every business organisation.

Take the case of an army, for instance. The rank and file under the orders of each corporal are at the same time subject to his control, which inhibits them from acting, and subject to his initiative which starts them into action. And as the rank and file to the corporal, so is the corporal to the sergeant, the sergeant to his immediate superior, and so on throughout the military hierarchy up to the commander-in-chief. And this is no mere fanciful or far-fetched analogy. It is a close and apposite illustration. For the whole military machinery may be actually regarded in the light of a nervous mechanism. It is by the direct action of their nerve-centres that the officers conceive and issue their orders to their subordinates, and it is by the action of their respective nervous systems that the subordinates receive these orders and carry them out. The analogy is closer still. For with orders or without orders the soldiers are continually under the influence of the military discipline, which inhibits

them from taking upon themselves any independent initiative, and which provides that on the completion of any active order they shall subside into an attitude of quiescent expectancy. This continuous condition of inhibition or control is from time to time broken by behests to activity, precisely as the similar continuous inhibition of the nerve-centres is similarly broken.

Again, just as the purely local stimulus of a mote on the eyeball is responded to by the purely local action of a blink of the eyelid, actuated by a single centre of the lowest rank, so the purely local stimulus of the single footstep is responded to by the purely local action of the sentinel's challenge, delivered by an individual of the lowest rank. The analogy may be carried into much further ramifications. Though no further action follow the blink of the lid, yet the occurrence of the tickling feeling proves that the stimulus was carried beyond the local and subordinate centre up to centres far higher in rank. And though no further action follows the challenge of the sentry if it be satisfactorily answered, yet the occurrence is reported and comes to the knowledge of far higher authorities. If the grain of dust is not dislodged by the blink, but remains as a cause of irritation, further action soon follows. The stimulus is irradiated from the local centre to centres higher and higher in rank, and is now intense enough to set these higher centres in action; so that the hands are brought to the help of the lids, and if they do not suffice to get rid of the intruder, the voice requests help, and the whole body moves towards the source of relief. Similarly, if the sentry's challenge does not suffice to arrest the intruder, and his attitude is menacing and indicative of support behind, the alarm given by the sentry to his superiors is followed by action on their part. The guard turns out, the regiment is aroused, supports are sent for, the whole army may be set in motion, and a general engagement may ensue.

The parallel may be pushed further yet. A minute mote falling on a normal eye causes a single blink, and the intruder is removed, the action ceases. But the same mote falling on an inflamed eye causes a much more lively stimulus and a much more vigorous reaction. The sensation instead of being insig-

nificant is acutely painful. The single blink is replaced by copious action of lids, and hands, and effusion of tears. As the inflamed eye to the normal eye, so is the army in the field to the army in time of peace. In the latter case the intrusion of a single stranger produces no reaction beyond what is necessary to keep him out, but the same occurrence in the face of the enemy causes a general commotion. No longer confined to the guard-room, the intelligence of the intrusion is conveyed to headquarters, and a general commotion results from the arrest of the spy.

The similarities are far from being yet exhausted. The private soldier has nothing to do with the supplies save only to take to himself the rations and clothing that are served out to him. Each subordinate officer is responsible for the condition of the clothing and accoutrements of those under his command. The colonel of the regiment, from whom proceed both the initiative to action for the whole regiment and the discipline which maintains the whole regiment ready to act, is further responsible for the supply of food, clothing, and arms for the whole of his regiment. The same combination of functions that the colonel fulfils towards his regiment the general fulfils for his brigade, the superior general for his division, and the commander-in-chief for the whole army. It is the same with the nerve-centres. Each of the lowest centres of all not only effects a movement, but at the same time governs the nutrition of the parts that affect that movement, so that when a motor nerve is cut, the muscle wastes. Each higher centre effects not only the control and the initiation of all centres beneath it, but their nutrition as well, and not only their nutrition, but also the nutrition of the mechanisms that they serve. So that while the lowest centres control the nutrition of (say) muscles only, the higher control the nutrition of the lower centres; and thereby, in a more general way, the nutrition of the muscles that the lower centres actuate, and thus control, moreover, the nutrition of bones, ligaments, &c., that are subsidiary to the movements that they directly affect. And as the commissariat is under the orders of the commander-in-chief, who governs through it the supply of nutriment to the whole army in general though not in detail, so the highest

centres regulate generally, but not in detail, the nutrition of the entire organism.

If the nervous system be constituted as is here supposed, then the parallel that is now suggested will hold good not only in health but in disease. Let us consider the analogous cases and see if it do so apply.

If the sentinel is killed, he can no longer issue his challenge to the intruder, nor communicate the alarm to headquarters. And if the reflex centre is destroyed, if, for instance, the frog is pithed, the usual stimulus provokes no reflex action, and no sensation corresponding to the stimulus is felt. On the other hand, suppose the sentry is excited by drink. He is met by the patrol, and in an excess of zeal he challenges and straightway bayonets his comrade. Similarly, if the frog be strychnised, a slight and ordinary stimulus produces an excessive and extraordinary reaction.

Suppose that it is the sergeant that is killed, which may be considered equivalent to having the cord bruised. The first consequence is that the rank and file receive no orders from their superiors, the sergeant who was the transmitter of the orders being killed. Consequently they remain inactive, but they are capable of again taking part in the military operations if the messages are sent by another route, or a new sergeant appointed and the communication restored. Similarly, when the cord is cut, the centres below are cut off from communication with the centres above, and no voluntary movement becomes possible. But the inferior centres are themselves intact, and are capable of resuming their part in the bodily operations if the cord recovers, and communication is restored. There will be a second consequence of the loss of the sergeant in the relaxation of discipline which he was instrumental in maintaining. His control being removed, the soldiers will be apt to get uproarious and exceed their duty. And when the control of the superior centres is removed by bruising of the cord the reflex centres overact, and reflex action becomes excessive. Further, as the sergeant is no longer present at the daily distribution of food, the interests of his men will probably suffer, and the amount served out to those who have been under his charge will be diminished. Similarly, when the

cord is bruised and the legs are paralysed, their nutrition begins immediately to suffer. At the end of twenty-four hours it is apparent that the muscles are flabbier, the skin looser and sweatier than normal.

Now let us suppose that the colonel is shot at the head of his regiment. For a short time the regiment is demoralised. It is thrown into disorder and begins to retreat. In a short time these effects pass off, and then it is found that the state of affairs is as follows: The severance of the chief channel of communication between the regiment and the commander-in-chief will greatly impair the value of the regiment to the army at large. The daily routine of the regiment will not be much affected. That is carried out by the subordinate regimental officers—by the majors, captains, &c.—and will be carried on in much the same way whether the colonel is present or no. The daily drills will be performed, the parades held, the canteen will be open, and the messes served as usual. The general knowledge of regimental duties attained by the inferior officer will suffice to carry the regiment through the simpler duties that are required of it on the march. But when there is a prospect of an engagement, when very special and precise and important and perhaps novel movements have to be carried out in the face of the enemy, then the loss of the guiding spirit is felt. When the skilful handling of the regiment, with reference to the particular nature of the ground and the presence and strength of the enemy, becomes of the first importance, then the want of a single, firm, decisive, experienced authority becomes patent. Compare these facts with the facts of paralysis from a small clot in the corpus striatum. Such a clot produces first a general shock with total paralysis of, say, the right arm. This corresponds with the initial retreat of the regiment and its virtual paralysis. When the effects of shock pass off, it is found that the arm is much weakened, and that it is weakened in such a way that the digits are altogether paralysed, the hand less so, and that the power of movement increases as we leave the periphery, while at the shoulder no defect of movements can be found. In other words, what are lost are the most peripheral movements; that is to say, the movements that are most precise,

most elaborate, most special, most important, and most novel; while the movements that are least affected are the most general, the most automatic, the simplest, and the most frequently repeated. The parallel holds good in all respects, and it may be carried further. For loss of the colonel involves the omission to send in requisitions for clothing and stores. The daily rations are distributed as usual, and the regiment does not suffer at once, but as the clothing and stores become worn out they are not replaced, and thus, after a time, the condition of the regiment suffers. So with the hemiplegic limb. At first little change in it is apparent, but after a time it wastes. The loss of the colonel will not have a very apparent effect on the discipline of the regiment, for that will be maintained in a general sense by the other regimental officers, nevertheless, it must be to some slight extent impaired; and correspondingly, while in hemiplegia, we do not find reflex actions very much increased, we do find the muscular and tendon reflexes slightly increased.

Suppose now, that in the excitement of battle the colonel loses his head. He begins to send out messages to his skirmishers faster and faster. No sooner is one order sent than another follows to contradict it, another and another and another follow as fast as the messengers can succeed one another. The consequence will be that the soldiers will run first to execute one order, then to execute the next, until, as different orders come, one after another, faster than they can be carried out, the soldiers will be raised to a state of high excitement; and after a good deal of aimless running to and fro, they will at length stand still and do nothing, and finally cease acting from exhaustion. The occurrence is parallel to a fit of Jacksonian epilepsy, which begins with the hand trying, as it were, to execute many movements at once, goes on to moveless rigidity, and ends in motionless exhaustion.

We will now transfer our observations to a higher level, and consider the result of the loss of the commander-in-chief. In actual life no doubt the loss would be supplied by sending out another commander-in-chief from home, but we must suppose not only the individual lost but the function suppressed. What will be the result? The result will be that while the

army maintains its general organisation intact, and while all the routine duties of regiment, brigade and division, of horse, foot and artillery are carried on much as usual, yet for campaigning purposes the army is useless. It ceases to be an assistance to its allies, a defence to its country, or a menace to its enemy. A civilian passing through its ranks and seeing everywhere the evidence of order and discipline, the trenches manned, and the sentries posted, would see no evidence of any important defect in the army. But although no defect might be seen by the civilian, the practised eye of a military observer would see that something was wrong. He would observe a languor, a want of initiative, a failure to take advantage of opportunities. Though the army might be marching in the right direction, its several divisions, perfect in their internal discipline, would not be so handled as to support one another at proper times and places. In case of an engagement, the whole army takes part, led by its generals of division, the soldiers fight gallantly, the guns are well served, the charges brilliantly delivered, one whole wing may be successful in driving back the enemy. But in the absence of a single guiding hand the result must be disastrous. The tactics may be admirable, but the strategy must fail. If there occur no general engagement, it may be long before the army is destroyed, but in the absence of a definite aim and plan, the inferior commanders become discouraged and lose their interest; the discouragement and lack of interest spread rapidly downwards from rank to rank until the whole army is disheartened. Then ensues desertion, cutting off of stragglers, failure of supplies; and soon dysentery sets in, and sooner or later the army melts away.

Corresponding to the commander-in-chief are the highest of all nervous centres—those centres whose function it is to actuate the movements and regulate the nutritions of the body as a whole, and whose activity is accompanied by consciousness. When these centres, and these alone, have their functions abolished, as in the early stages of drunkenness, and of other forms of coma, what do we find?

To the superficial observer and the layman, who are comparable with the civilian in the example, the difference is not

great. As in the one case the local and routine duties are efficiently performed, so in the other are the local and vegetative functions. As in the one case no part of the army is perceptibly defective, so in the other no part of the body is conspicuously paralysed. The languor, the want of initiative, the failure to take advantage of opportunities on the part of the army is paralleled by the heaviness, the dulness, the defect of intelligence on the part of the individual. It may happen that the divisional commanders of the army are actuated by mutual jealousies and suspicions, and that when the controlling hand of the superior is removed they fall out, quarrel, attack one another, and diffuse throughout their several divisions the excitement which they themselves display. Or it may happen that, their ardent spirits no longer held in check by the wisdom and authority of their chief, they launch out into reckless enterprises, in which their troops become speedily exhausted or meet with disaster. Similarly, when the highest nervous centres are thrown out of gear, the most prominent symptoms of the loss may be an outbreak of violence or a clamorous exaltation, and this is due, it may be contended, to the uncontrolled action of centres immediately subordinate to those whose function has been lost. Such occurrences are common in the early stages of drunkenness, of insanity, and of other forms of coma.

But whether the immediate effect of the loss of the highest authority is an outbreak of excessive but misdirected vigour, or whether it is a general condition of diminished activity, in either case the ultimate result is a loss of efficiency on behalf of the body at large. We have seen how in the army the routine of discipline may be retained and the routine operations carried on, and that in the body the vegetative functions are preserved, but in both cases the highest functions which are necessary for the conservation of the organism as a whole, are lost. The march of the army is continued, but when it arrives on the field of battle the want of a coherent plan of operations loses the day. Similarly the individual in an early stage of drunkenness is competent to get to his office, but when he is there he is unable to transact business efficiently. The army without a chief becomes a *quantité négligeable* both to

friends and foes; and similarly the man who is indulging in a drunken debauch is useless. Although no one part of the army is actually disorganised, and no one limb of the body is paralysed, yet throughout the whole of each body there is a languor, an inability for sustained exertion, an impaired efficiency, which, considered from the present point of view, may be regarded as a paresis of the entire organism. It is not conspicuous in any one member, because, all being equally affected, the emphasis of contrast is lacking; but if we compare the organism as a whole in its present condition with itself in its former condition, or with other similar organisms that are unaffected, it becomes at once apparent that there is a universal defect of efficiency, which in both organisations may be ascribed to the withdrawal of the stimulus of frequent positive impulses to different and new exertion—a withdrawal which is followed in both organisations by disheartening, discouragement, and diminished inclination to exertion.

The lack of initiative, the feebleness, and the possible uproar from absence of the controlling authority will be the most conspicuous, but they will not be the only effects of the removal of the highest functions either in the one organisation or in the other. In the army the supply of arms, ammunition, accoutrements, clothing, stores, and food depends on the requisitions sent home to the War Office by the commander-in-chief. If the commander-in-chief ceases to exist the stores will still be sent out, no doubt, but they will be sent in wrong proportions, haphazard and by guesswork—too much of this and too little of that. Arrived at the army, the commissariat officers sharing in the general inefficiency and carelessness, the distribution will be similarly faulty. Here will be carbine ammunition served out to infantry regiments, there will be cavalry soldiers with shoes instead of boots; in another place, horse-shoes provided for a camel regiment. The distribution of food too will be badly done. The consequence will be that the army will suffer, not only in efficiency, but in physique. The men, being badly shod, badly clad, and badly fed, will soon deteriorate in health. In the individual, too, loss of the highest function invariably causes mal-nutrition. In drunkenness and in insanity the

skin becomes altered in tinge, becomes unduly dry or unduly moist; the hair is harsh and staring, or less often dripping with moisture, the nails are affected, the bowels are constipated, the breathing, the pulse, the urine are altered. To sum up, the three main consequences of loss of the commander of an army are lack of initiative, lack of control, and modification with impairment of supplies. And the three main consequences of loss of the highest nerve-centres are lack of initiative, exaggerated action, which is a consequence of lack of control, and modification with impairment of nutrition. Here, therefore, the parallel between the military organisation and the organisation of the nerve-centres still holds good. It still remains to compare the opposite error, the fault of over-action, and see if the same holds good.

Suppose the commander-in-chief to become highly excited, and to issue orders in great excess. The result will be an excess of activity on the part of the army at large, but will vary with the degree to which the orders are excessive, and with the directions in which they are issued. A multitude of contrary orders following one another faster than they can be executed will have the same effect when issued by the general to the whole army as when issued by the colonel to his regiment. They will produce a great excess of activity culminating in a standstill of consternation. The various movements will interfere with one another, so that while every man is put to far more exertion than any one movement requires, the practical result will be *nil*. The analogous process in the individual is a universal convulsion.

To recapitulate.

The propositions that I beg to submit for the consideration of the society are these:—

1. Every nervous discharge takes place normally in the face of a continuous resistance.

2. Every nervous centre is normally subject to a continuous control or inhibition.

3. This inhibition is not exercised by any separate portion of gray matter, but concurrently with other functions.

4. Every destruction of nerve tissue produces a double effect: a negative and a superpositive.

5. The superpositive effect can be produced only by the destruction of an inhibition.

6. *Ergo*, every nerve-centre not only is subject to, but exercises, an inhibitory influence.

7. Every nerve-centre controls simpler centres, and is controlled by more complex centres than itself; so that—

8. The nerve-centres are arranged in a hierarchy.

DISCUSSION.

Dr. Hughlings-Jackson.

After remarking on the great value of Dr. Mercier's paper in many important ways, Dr. Hughlings-Jackson said:—

I quite agree with Dr. Mercier in the principle of a hierarchy of nervous centres. I have suggested a division of the central nervous system into three levels. (See paper, 'Journal of Mental Science,' April 1887, section 5.) The relations of these centres to one another is a very complex one. Besides relation of direction and control, the higher their evolution the more independent do centres become in their activity; there is quasi-detachment, so that in the most "detached" (the highest) centres activities can go on (internal evolution) largely independent of present converse with the environment; on the psychical side there can arise trains of thought independent of present experiences (I have considered this aspect of the subject in the paper above referred to, 'Journal of Mental Science,' section 8).[1]

I think it would be best to use the term "force" as a name for the rate of liberation of energy by nervous elements (nervous discharges). It seems to me that rate of liberations of energy by nerve cells, or as I shall say, rate of discharges, is a thing of great importance for our consideration with regard to the interpretation of some of the phenomena of epileptiform and epileptic seizures, and of their after-conditions. The bearing is particularly on ranges of convulsion, on sequence of convulsion, and ultimately on the interpretation of certain negative and positive symptoms found when the fits are over (after-conditions). It is with regard to the nature of these after-conditions that we are mainly concerned with inhibition; we are concerned at the same time, in principle,

[1] See Spencer, 'Princ. of Psych.,' vol. i., chap. 6, footnote, and especially Section 264.

with one of the factors in all cases of insanity. I remark first on the fits.

I take for illustration the comparatively simple epileptiform seizures, dealing with that variety in which the initial convulsion is of the thumb. In such a case there is a very local hyper-physiological[1] state of some nerve-cells of a part of the Rolandic region (so-called motor region)—a local "discharging lesion," or, as I sometimes call it, a "physiological fulminate." It is well known that there are many ranges of convulsion in such a case, from that seemingly limited to the thumb, to universal convulsion. With regard to ranges we have not only to consider the number of cells highly over-unstable in the local "discharging lesion," but their degree of instability—not only the different quantities of "explosive" matter making up physiological fulminate, but the different degrees of "explosiveness" of that matter. Supposing a quantity of gunpowder and a quantity of gun-cotton (each having the same quantity of energy), the effects of the explosion of the two would be very different, the latter exploding so very much more rapidly. I submit that *one*[2] factor governing range of convulsion is rate of the discharge; I speak now of the primary discharge, that is, of the discharge of the cells of the "discharging lesions."

We shall speak of rapid and slow discharges in epileptiform seizures. All discharges producing paroxysms of convulsions are very rapid in comparison with the discharges in health. But the abnormally rapid discharges producing convulsion differ in rate; they are comparatively with one, another, rapid and slow. I fear "rapid" and "slow" are not the right words. I do not mean that "nerve impulses," after discharges which produce convulsion, "travel" faster than those after the discharges of health; but that in the former case more numerous "nerve-impulses" are emitted in a given time, or else that more cells are simultaneously discharging upon the same part, "a greater quantity of motion"

[1] The pathological processes (disorder of nutritive process) leading to the local hyper-physiological state does not concern us here.

[2] I say one factor, because we have to consider the number of cells highly over-unstable as well as degrees of high over-instability of them. Further, in paroxysms in cases of epilepsy proper, the "discharging lesion" (highest centres) will be of cells of nervous arrangements which are highly integrated, that is, nervous arrangements (or units) which represent great ranges of the body in very intricate combinations. In the case of epileptiform seizures (middle motor centres) they will be all of less integrated nervous arrangements. The discharge of the higher centres will, on account of the higher degree of evolution (factor integration) of those centres, produce wider and more simultaneous effects.

in a given time. (The reverse for "slow discharges.") If the primary discharge in a fit be slow, slow in the sense I mean, the "currents developed will "flow" more exclusively to that part which is most specially represented by the cells of the nervous arrangements of the "discharging lesion;" in the case chosen for illustration, say, to the thumb only. If the primary discharge be rapid (the same quantity of energy being liberated as in the former case, that of slow discharge), the currents will "flow" also to parts less specially represented by cells of those nervous arrangements, not only to the thumb, but to other digits also; the convulsion will be of greater range. To say that a part of the body is most specially represented in a centre, means that it is united by lines of least resistance to that centre, the parts less specially represented in it being united to it by lines of greater resistance. Let us arbitrarily suppose three degrees of resistance between the discharging lesion and three parts of a muscular region it represents; (1) least, (2) more, (3) most. A very slow discharge may be limited to (1), a more rapid to (1) and (2), and a most rapid will involve all three parts. We are supposing that the same quantity of energy is liberated in the three instances, urging that the more rapid liberations overcome greater resistances, and thus that there is wider spreading of effects on the periphery. The more rapid the discharge, the more nearly simultaneous will the convulsion of the three parts be; the less obvious the sequence, the more nearly equal will be the degree of convulsion of each, and the sooner over will be the attack; speaking very roughly indeed, the effects of a more rapid discharge will be spread out more widely, more thinly, and will last for a shorter time. The slower the discharge, the more of a decided sequence will be the convulsion, the less equally will be the degrees of convulsion of each part and the more lengthy the attack; the part first involved will suffer most. No doubt in all cases there is a *compound sequence*, but in very rapid convulsion we may not trace it; in slow convulsion the compound sequence may be traced and symbolised as x, then $x^2 + y$, and then $x^3 + y^2 + z$.[1]

I have so far spoken as if the discharge were primary only, that

[1] Nothing beyond rudest quantification is of course meant by these symbolisations, in spite of the employment of definite indices. I have often urged that we should pay attention to compound order of increase of paralysis, dependent on increasing gravity of destructive lesions, and to compound order of spreading of convulsion dependent on increasing discharges. Nervous evolution does not imply insensible gradations, but increases in compound order. I suppose that increase in compound order, as roughly symbolised in the text, applies to all increasing activities of the nervous system (including those correlative with mental states) both in health and disease.

is, as if it were of cells of the "discharging lesion" only; possibly it may be so in very slightest local seizures, as when the spasm is of the thumb and scarcely at all of other parts near to it also. But when a convulsion starting in the thumbs spreads greatly in range (say to the arm, face and leg, or further still), the supposition is that the primary discharge, that of the fulminate (otherwise "discharging lesion"), upsets the equilibrium of, that is discharges, *normal* cells, with which the cells of the fulminate are connected, and compels them to "co-operate in its excess"; there are primary and induced secondary discharges. But it is supposed that the rate of the primary discharge always governs the situation —that rapid discharges lead to wider-spread, to more equal convulsion of the parts engaged, to more temporary convulsion and to convulsion of less distinct sequence; whilst slow discharges lead to more local, more unequal convulsions of longer duration and more decided, more traceable sequence. (The same quantity of energy is supposed to be primarily liberated both in the rapid and slow discharges (by the "discharging lesion").)

I now consider post-paroxysmal states. After epileptiform seizures there is paralysis. I submit that after slow discharges (slight fits long continued and limited in range), the paralysis is very local and decided, and therefore obvious; after rapid discharges (severer shorter fits) it is wide-spread, "diffuse," and therefore easily ignored. In the former case it lasts longer than in the latter. I submit that in the former case there is really less paralysis than in the latter. This, however, is denied: the very contrary is affirmed. As an objection against an hypothesis I contend for (essentially Todd and Robertson's). The assertion has been made that there is more paralysis after slight than after severe fits. It is said that whilst after slight epileptiform seizures there is decided paralysis, say, of one arm, there is none after the severest epileptic seizures; my contention is that the whole physical condition in the latter is paralytic, and that there is very much more paralysis than in the former case.

Applying Todd and Robertson's hypothesis to post-epileptiform states, and bearing in mind what has been said as to the difference in the effects of slow and rapid primary discharges (of the "discharging lesion"), and consequently in the secondary discharges (of normal elements), I submit that after slow discharges the post-epileptiform exhaustion of central nervous elements is limited in extent, very local, very decided in degree, and that it remains for a comparatively long time; whilst after rapid discharges the exhaustion is wide, diffuse, slight in degree, and remains for a short time.

I now apply this generalisation to after-conditions of paroxysms of epilepsy proper, and thus come closer to the object of Dr. Mercier's address. We have here to do with the highest cerebral centres—popularly "mental centres"—epileptic paroxysms being owing to "discharging lesions" of small parts of these centres. These after-conditions are, scientifically regarded, cases of insanity; what is called epileptic mania is one example. Epileptic mania should be called "post-epileptic 'loss' of consciousness" (a purely negative mental state), with "mania," (a diametrically opposite, a super-positive mental state). Post-epileptic coma is certainly an insanity; it is acute temporary dementia (see Dr. Mercier's admirable contribution to the realistic study of insanity, a paper entitled *Coma* ('BRAIN,' January 1887, especially p. 483). In all other cases of insanity, the condition is essentially similarly duplex; there is in every one some degree of negative affection of consciousness, and in every one there are also some super-positive or positive mental manifestations, except in absolute dementia, if there be such a thing, when the mental condition is entirely negative. Of course our proper concern, as physicians, is not with negative affections of consciousness, but with the physical states they imply. I submit that, taking the case of epileptic mania, the "loss" of consciousness answers to what is physically exhaustion (functional absence) of elements of the highest "layers" of the highest centres (Dissolution), and that the mania answers to activity of the next lower "layers" and rest of the nervous system (the mania signifies the Evolution going on in what is left intact of a maimed nervous system). But the further supposition is that, the higher "layers" of the highest centres being functionally absent, the next lower layers are uninhibited, and are in consequence in greater activity than normal. Before saying more on this point I would clear the ground by stating what I think are the four factors in post-epileptic and in all other cases of insanity.

The factors are: (1) Different depths of Dissolution of the highest centres and consequently different heights of Evolution remaining.[1] (2) The rate with which the Dissolution is affected. (3) The nervous system in which the Dissolution occurs (the person, young or old, clever intelligent, timid or brave, &c. &c.). (4) The influence of external conditions and of local bodily states.[2] I shall consider only the second factor.

[1] If we distinguish between uniform and local Dissolutions of the highest centres, there would be five factors in cases of insanity.

[2] I have been misunderstood to have put forward the hypothesis of Evolution as a basis for the clinical classification of insanity. For clinical purposes we must have arrangements of them by type, for a long time to come at any rate.

I submit that the more rapidly Dissolution is effected in cases of insanity (the more rapidly the higher layers of the highest centres lose function), and thus the more quickly control is removed (from lower "layers"), the greater is the rate of activity on the lower level of Evolution remaining, and correspondingly the more super-positive are the positive mental symptoms. If we take extreme cases, we see that the senile dement whose Dissolution is effected with very great slowness is quiet; whereas the post-epileptic maniac, who has undergone Dissolution some thousand times more rapidly, is the most furious of all maniacs (activity at a very great rate is what here concerns us). I wish to show the bearings of what was said on different rates of discharges in epileptic fits, and on the varying condition of the highest centres after them, on the symptoms of post-epileptic insanity. (I am still neglecting three out of the four factors in insanity.)

In all cases of post-epileptic insanity Dissolution has been rapidly effected, but there are degrees here; in some it is comparatively slowly, in some comparatively rapidly effected. Clinically there are after some very slight epileptic fits during "unconsciousness" exceedingly elaborate[1] actions, which to bystanders may seem nearly, if not quite normal. These actions are also quiet (at no great rate), and are long continued. The post-epileptic condition has, in these instances, the three characters (i.) great complexity, &c., of actions at but (ii.) little rate (busy, perhaps, but not rapid), and (iii.) lengthiness. I submit that the discharge in the prior fit was slow, and consequently that the exhaustion in the highest centres effected by it, remaining when that discharge had ceased (Dissolution), is very limited in extent, very local, very slowly effected, and very decided in degree; consequently the lower level of evolution remaining in those centres is high, the activity on that level is at a slow rate and is long continued. Supposing that there are "all degrees" (which here I neither deny nor affirm) from such cases to cases of furious post-epileptic unconsciousness with mania, I will take the last cases in further illustration. I submit that in these the exhaustion in the highest centres after the fits (Dissolution) is very wide, "diffuse," was rapidly effected and is very slight in degree,[2] and that the

[1] We have nothing to do here with the "importance" of the actions after epileptic fits, but with their degrees of complexity, speciality, &c.

[2] Not being an Alienist physician, my experience of these degrees of post-epileptic states is small. I deal with cases I see, and speak subject to correction by those whose experience of what are called mental diseases is greater than mine. I by no means pretend to explain the various ways in which temporary insanity is associated with epileptic fits.

level of Evolution is low, that the activity on it is at a great rate and of short duration.

I have nothing to say on the nature of the inhibitory process. I would urge, however, that here, as in all other cases, we should distinguish between the psychical and the physical; we cannot legitimately speak of any mental states as inhibitory of any physical processes, any more than we can speak of them as producing movements. I have long urged that controlling as well as directive action is displayed throughout the nervous system.[1]

It is quite certain that we have in some way super-positive effects consequent on negative lesions; indirectly consequent, of course. I have illustrated this dogma sufficiently in one way in the preceding remarks, and have considered it with regard to the temporary insanity produced by alcohol and the insanity of starvation in the "Croonian Lectures" ('Brit. Med. Journal,' April 12th 1884). The dogma is, I think, applicable in cases where questions of inhibition do not arise. "Disorders of co-ordination" in cases of negative lesions are, I submit, cases of paralysis; the negative lesion, that caused by the pathological process, causes paralysis only, paralysis in the sense of loss of some movements of the regions affected (the most special); what is called the disorder of co-ordination is the "forcing" of (over-development of) other movements of the same region (the more general) by activity of healthy nervous elements, in the fruitless endeavour to develop those no longer possible. If, in the case of the ataxy of tabes, the negative central lesion does not cause loss of movements by destroying any motor nervous arrangements, there is nevertheless (obviously in an early stage of this "disorder of co-ordination,") underdoing of some movements and overdoing of some others. In an early stage we see defect in the movement in which the peroneus longus is chiefly concerned, and overdoing

[1] The following is from my second "Croonian Lecture," 'Brit. Med. Journ.,' April 5, 1884: "Roughly, we say, that there is a gradual 'adding on' of the more and more special, a continual adding on of new organisations. But this 'adding on' is at the same time a 'keeping down.' The higher nervous arrangements evolved out of the lower keep down those lower, just as a government evolved out of a nation controls as well as directs that nation. If this be the process of Evolution, then the reverse process of Dissolution is not only 'a taking off' of the higher, but is at the very same time a 'letting go' of the lower. If the governing body of this country were destroyed suddenly, we should have two causes for lamentation; (1) The loss of services of eminent men: and (2) the anarchy of the now uncontrolled people. The loss of the governing body answers to the Dissolution in our patient (the exhaustion of the highest two 'layers' of his highest centres); the anarchy answers to the no longer controlled activity of the next lower level of evolution (third 'layer')."

of that in which the tibialis anticus is chiefly concerned. (I have considered this matter in a paper, "Journal of Mental Science," April 1887, Section 10.)

Dr. Francis Warner.

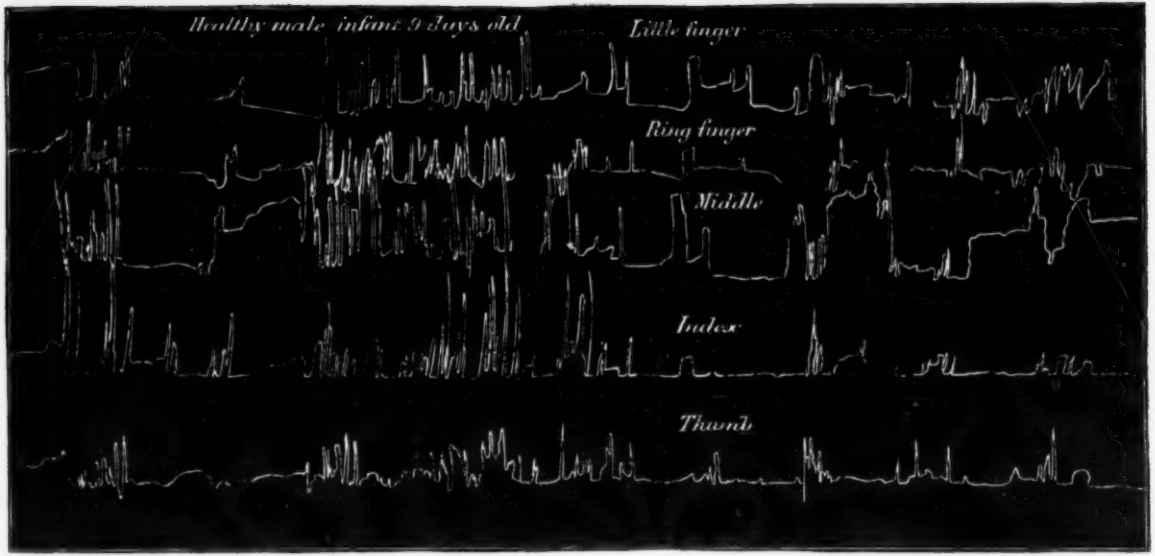
The study of Inhibition is so wide a subject, that I propose to confine my remarks to cases where motor action of the brain stops sequent to an impression received through the ear and the eye. It is proposed to consider observations as to what may be seen sequent to the period of inhibition; together with inferences as to what occurs during the period of no outward display of action in the brain.

Tracing 1 (see Fig., p. 394) shows spontaneous movements of an infant during fifteen minutes; the method employed is described in the 'Journal of Physiology,' vol. iv. No. 2.

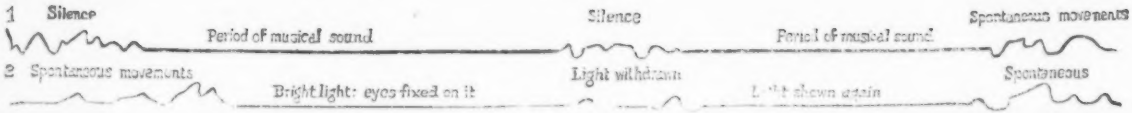
Tracing 2 shows the spontaneous movement of an infant five months old; this is seen to be inhibited by the sound of a musical-box, or by a strong light. The effect of the sound, or the light, was to arrest or inhibit movement temporarily; our chief concern is to study what occurs in the brain-centres during the time of no movement, *i.e.* during the time when no efferent currents are passing from the centres to the muscles.

I will not stop here to argue that light or sound, acting upon the eye or ear respectively, do produce a physical impression on the nerve-centres. I have elsewhere enunciated the proposition that "every vital act requires among its antecedents a supply of pabulum and stimulation by some force;" having previously given evidence in support of that proposition, I will not repeat it here. I have also shown that an impression produced upon a nerve-centre or group of centres may be delayed in its expression, and known to us only by subsequent series of movements. I now proceed to suggest how we may observe the results of what occurred in the centres during the quiet time when no efferent currents were passing from it. Common experience shows that after exercising the child frequently, say twice a day for fifty days, "he has learnt to know the musical-box," and this is indicated by the exact repetition of a particular series of movements, or efferent currents proceeding from certain nerve-centres to the muscles following the sight or sound of the box. A getting ready for action among the centres resulted during the one hundred periods of inhibition.

Now for a second example. Observing a school during a drilling lesson, I saw the first, second, and third classes go through the same exercises to the same words of command. The first class had



TRACING 1.



TRACING 2.

been similarly drilled, say, one hundred times, the second class sixty times, the third class only twenty times. In the first class the exercises followed in their detailed series of nerve-muscular movements with great uniformity, exact in the time and quantity of movement in each part, and each group of nerve-centres. The command is followed quickly and surely, by the particular series of movements. The exercise has been well learnt by this first class; it is less exact in the second class. The third class did not "know" the exercises, they (*i.e.* their nerve-centres) had not been taught sufficiently; they must be further impressed through eye and ear before their exercises become as exact as those of the first class. The children being required to stand in order motionless, and unimpressed by other sights than the teacher, they see the exercises as he performs them (sight impressions), then at the word of command (sound impressions) they (*i.e.* these centres) perform the same or similar series of movements.[1]

Returning to what took place in the baby's brain, after one hundred impressions by the light, and the sound of the musical-box, we find his centres tending to act in groups, as indicated by definite groups and series of movements. It may be inferred that some kind of change has been produced among the nerve-centres, forming them into functional groups, or groups functionally connected for action. Some explanation of the hypothesis of functional unions of centres must now be given. When a definite combination of movements of certain parts (*i.e.* combination of centres sending efferent currents) follows with uniform exactness upon a certain stimulus, it may be assumed as a working hypothesis that those centres have in some way—I do not know how—become "tied," or functionally united for action. If a stimulus, N, is uniformly followed by movement of the parts A, B, C, D, it is assumed that the centres a, b, c, d, coresponding, are in some way tied, or compelled to act together. It would commonly be said, the more uniform the recurrence of the action the more automatic it is. During the period of inhibition the centre is being stimulated its nutrition is probably as high as at other times, but no efferent currents are passing from it to the muscles, there is no expenditure of force in that way, the nerve-force is being spent in making ties or special unions. Observation of what follows shows us what has been occurring during inhibition; functional unions have been formed and the centre got ready for combined action—work was being done during the quiescent stage. Observation of the delayed expression of the impression produced during inhi-

[1] See 'Anatomy of Movements,' p. 77.

bition, affords evidence that functional ties or unions of centres were formed.

In the third class at the drilling-lesson, unions among nerve-centres are formed, while the children are quiet and looking at the teacher; the efferent expression of those unions is seen in subsequent movement on the command being given.

In this union of nerve-centres, while sending no efferent currents to muscles, I believe that we have the representation of some forms of brain action concerned in producing thoughts, or rather the brain state corresponding thereto. There seems to me no evidence that the period of inhibition in the cases given is a period of no action in the nerve-centres, but rather that it corresponds to the molecular changes which represent the mental state called "attention," or "getting ready for combined action among nerve-cells," and the physical process of "learning." All expression of mind in action is by movement, and the expression "unions of cells" may be delayed indefinitely, not appearing till some fresh stimulus, such as the word of command or some object seen, stimulates efferent currents from such groups of cells expressed by series of movements.

Dr. W. H. Broadbent.

I think Dr. Mercier will find the Society prepared to go with him a long way in the direction of the idea of Inhibition which he has brought before it. For my own part, I have never accepted the hypothesis of special inhibitory centres, but have looked upon inhibition as a simple corollary of the action of one nerve-centre upon another—command implies control. It has, indeed, seemed to me that the mystery surrounding the question of inhibitory influence, of which Dr. Mercier speaks, is very much of our own making, and is a mystification rather than a mystery.

The simile of an army, which he employs, is one which must have occurred to many minds, and I have some recollection of its use before as an illustration of the subordination of lower to higher centres in the nervous system; but he makes a new and ingenious development of it, in order to get rid of the necessity for special trophic centres and nerves, distinct from other centres and nerves, in which conclusion I agree with him, although it would be easy from the same comparison to argue the reverse.

I cannot, however, accept the idea, on which so much of Dr. Mercier's reasoning turns, that there is any necessary inherent tendency of a nerve-centre which has begun to discharge to go on discharging until it is utterly exhausted. There is just as much

reason to assume, that a muscle once called into action must continue to contract till its store of energy is expended, for, like a nerve cell, it is a reservoir of force. All nervous action is in response to some stimulus, and it lasts only so long as the stimulus which calls it forth continues. When light falls upon the eye, or sound upon the ear, a series of nerve action or discharges is set up, which ceases with the cessation of the visual or auditory impression, and, in like manner, central discharges down the motor-tract are conditioned by some determining influence. A nervous discharge may be compared to the ringing of the electric bell, which begins and ends with the pressure on a button, which, by making and breaking the circuits, conditions the evolution of stored-up energy.

A minor criticism is a repetition of that already made by Dr. Hughlings-Jackson, in which he points out that the tremor of paralysis agitans and the tremor of drunkenness, or of exhaustion, are of a totally different kind, and cannot possibly illustrate the same condition. Like Dr. Jackson, I have an hypothesis as to the seat of the morbid change in paralysis agitans, which, emboldened by his example, I venture to mention; I believe the lesion to affect the muscular nerve endings. I may, perhaps, be permitted to add also, that the few epileptic fits which I have happened to see, have not ended in the terrific universal spasm which he describes, but more frequently in a series of general spasmodic jerks of diminishing intensity.

The idea as to the nature of nerve energy or nerve force, which underlies Dr. Mercier's reasoning on the subject of inhibition, and which is generally accepted, is that each cell is a reservoir in which force is stored, and each fibre a channel by which force is carried; the whole conception is one of liberation and transmission of energy. An impression on a sense organ sends a wave of force up to the centres; a motor impulse is an explosion in certain cells of the cortex, and the energy set free travels downwards till it impinges upon given muscles and calls them into action. I have for a long time held a different hypothesis of the nature of nerve force, and of the relation in which the nerve centres stand towards each other and towards the peripheral nerve-endings. This is that the different centres among themselves, and the centres and the peripheral terminals of the nerves in muscle or sentient surfaces, hold each other in mutual tension, the periphery reacting on the centres in immediate relation with it, and the lower upon the higher centres, as well as the higher upon the lower and upon the periphery, and that a nervous impulse is not so much a transmission of energy as a disturbance of

equilibrium. The lower centres and motor nerves and muscles do not passively await the shock of the explosion in cortical or other cells, but are constantly in touch with these cells by a sort of back pressure. The best illustration of the mode of action which I believe to obtain throughout the nervous system, is furnished by telegraphy. When a message is sent across the Atlantic, it is not a flash of electricity which starts from the battery here, travels along the wire and affects the needle at the other side. There is a battery at each end, and the connecting wire is maintained in a condition of electrical tension or charge. What happens in the transmission of a signal is that disturbance of equilibrium at one end determines a corresponding disturbance of equilibrium at the other. It is in virtue of the susceptibility, due to this state of charge in the connecting wires, that multiple and opposite messages can be transmitted at the same time along the same wire. In the same way it is not force generated or liberated in the cells of the cortex, and transmitted to the muscles which sets them in action, but the entire chain of centres and the nerve terminals being in a state of tension or charge, energy liberated in certain cortical cells disturbs the equilibrium of corresponding lower centres, and ultimately of the muscular terminals, which last, as by the pulling of a trigger, set free the latent energy of the muscular fibres. To employ another more crude and less exact analogy, the difference between the received view of nerve-action and that which I am attempting to set forth, may be compared with the transmissions of energy by means of water dashing down an open channel and through a closed tube.

This idea of the relation between the nerve-centres and the periphery, in which, not the centres only, but the nerve-endings and the structures in which they are distributed exercise the office of batteries, which maintain the connecting nerve-fibres in a state of charge, seems to me to render more easy of comprehension various phenomena normally exhibited by the nervous system, and it may, when more fully worked out, throw light on morbid phenomena. For example, it gives to the peripheral end-organs an importance contended for by many investigators and thinkers, but generally ignored. Ganglionic powers, it is true, are conceded to the retina and to the expansion of the auditory nerve, scarcely inferior to those of the cerebral cortex, but corresponding powers must belong to the cutaneous nerve-endings. Muscle, again, has been said to be only less nervous than nerve; on the view here contended for, this follows naturally, and muscular tonus is an expansion of the tension assumed to exist.

Again, the degree of tension maintained throughout the nervous system may be high or low. When it is low, there will be diminished stability, and, as a result, the effects which Dr. Warner has described and represented graphically before the Society.

In telegraphy the equilibrium may be disturbed either in a positive or in a negative direction, and it is not impossible that some parallel to this may occur in the nervous system. It would follow, too, on the hypothesis of mutual resistance of the centres among themselves, upwards as well as downwards, on both the motor and sensory sides of the nervous system, that sudden withdrawal of this resistance on the part of a lower centre might determine discharge in a higher centre; in this way, epileptiform convulsions might have their starting-point in the basal ganglia or in the cord.

So far the illustrations are taken chiefly from the motor side of the nervous system, but, of course, the relation of tension will exist between the sensory and motor sides of the nervous system, between the sensory and motor nerve-nuclei at the same level in the cord, which are concerned in simple reflex action, between the basal sensory and motor ganglia, and between associated sensory and motor areas in the cortex; between the two hemispheres, and between the cerebrum and cerebellum, and between these again and the cord.

Inhibition is a simple corollary of the relation here supposed to exist between the different parts of the nervous system. The increased knee-jerk induced by pulling the hands is an expression of the intensified general tonus, and the inhibition of reflex and other movements by powerful impressions on sensory nerves, which finds no place in Dr. Mercier's theory, is explained by the heightened tension thereby induced, primarily in the related centres, and then throughout the nervous system.

Trophic changes seem to me to be more comprehensible when the importance of the nerve terminals is recognised, and their relations with the tissues are considered. The ultimate ramification of the nerves, in the structures to which they are distributed, are so completely lost in these structures that they may be said to form part of them, unless, indeed, we say that the structures, especially such structures as skin and muscle, are the nerve terminals, instead of saying that they receive them; they have everything in common, and whatever affects the nutrition of the one affects that of the other. On the one hand, then, the state of nutrition of the tissues will determine the degree of tension maintained in the periphery of the nervous system, and, on the other, the tension prevailing in the nerves will influence the

nutritive operations in the tissues. It is interesting to note that, in the most familiar trophic derangement of nutrition, Herpes Zoster, the disturbing influence travels in the opposite direction to the normal functional impressions in the nerves affected, which are sensory and centripetal.

Another recommendation, which the view of sustained charge and mutual tension of the nerve-centres and periphery, which I have attempted to sketch, has for my mind, is that it renders somewhat less incomprehensible the extraordinary phenomena seen in hysterical women, which Charcot has brought to our knowledge. Physiology, as yet, has scarcely taken them into account, and in nervous pathology they stand apart, and are not correlated with other diseases. A distinguished friend of mine indeed once went so far as to say that woman ought to be excluded from the domain of nervous pathology. In my opinion, on the other hand, the physiology and pathology of the nervous system will have to be rewritten from the point of view opened out by these phenomena. There is no structural difference between the nervous system of a hysterical subject and that of a healthy man, and the hemi-anæsthesia with its transference from one half of the body to the other, and the hypnotic phenomena, betray tendencies and reveal potentialities and relations dormant in the healthy nervous system. Now, if all parts of the nervous system are in a condition of charge, and hold each other in mutual tension, the transference of hemi-anæsthesia becomes in a way comprehensible; and the comparison which makes hypnotism a state of polarisation of the nervous system is in some measure justifiable.

Nothing could be easier than to find objections to the view here crudely set forth. At present, however, it is simply a tentative hypothesis, and is placed as such before the Society, and it would be premature to state and discuss the difficulties which suggest themselves.

DR. SAVAGE.

I wish to ask Dr. Mercier if he makes any distinction between Control and Inhibition?

I confess that I rise with diffidence, because I do not yet fully know what this inhibition is: if it is only control, then I do know something about it; but if not, then I shall only expose my ignorance in speaking.

I can understand control and moral control, or self-control, but moral inhibition I cannot grasp as a thing apart.

As to control, it seems the whole matter is part of our very existence, we learn control in one way or another or we perish. The facts of control are the facts of developing in our environment. We are moulded or controlled by our surroundings.

Dr. Warner has pointed out that the infant is in a state of uncontrolled restlessness. Quite so; it is learning what it can and what it cannot do, and those children who never learn, become idiots or perish; they have no power of control, which comes to this, they have no power to store any energy.

At the other end of the scale I see the persons in Asylums who have lost this power of control, and as a result fall out of relationship with their surroundings.

All theories and hypotheses have to run a pretty severe fire before they are accepted, and with Dr. Mercier's there will be the same experience, for though it covers many wants it does not comply with all. I do not understand the fluid, or whatever it is, which is manufactured, stored and distributed through the nervous system, for among the insane I meet with very surprising cases, if they are to be explained on the fluid theory. Thus I remember a patient who having had a very severe series of fits affecting one half of the body, passed into post-epileptic stupor. This lasted but a short time, during which she was hemiplegic; she awoke in a state of acute mania, using all her limbs with great vigour. This stage lasted some hours, when again hemiplegia was present, and this lasted for some weeks. Several similar attacks occurred before the death of the patient, and no coarse lesion was discovered. That any theory of storage and expenditure will explain this, I doubt; but I do not think that the suggestion of Dr. Broadbent, that the communication along the nervous lines depends on tensions, is much more likely to be true, and may be made to explain this and similar cases.

I do not agree with all the facts of Dr. Mercier. Thus, when he says that if we "have tried hard to stifle a laugh or yawn, and if it breaks out in spite of the inhibition, it is more violent than if we had at first given way to it; and the same is surely true of an outbreak of temper." I can only say that this last appears to me to be a very unchristian doctrine, and would not tend to the good of society, as no one would be at the risk of checking his little tempers lest they became horrid rages. We are very much dependent on hypotheses for the development of our knowledge, and we depend greatly on our hypotheses by the use of similes. Some object to this, but it is the old and recognised plan to explain the unknown by reference to the known, and the more we study nature the more are we forced to believe that one set of laws governs

every action; if we only get at the right set of laws, then we have nothing to do but fit them. The parallel given by Dr. Mercier of the hierarchy of control as seen in the army is very pretty. As far as the serial arrangement is concerned this is correct; but it will not do to push likeness too far, for we are taught that the highest nervous controlling parts are the latest developed and the least stable; whereas in the hierarchy of command in the army, surely the Commander-in-Chief is not the youngest and latest development, nor is he the least stable.

The paper is one which has made us think, and we have reason to be grateful to the author.

DR. CATTELL.

It may be worth our while to spend a few minutes in considering Inhibition from the mental or psychological side. The subjects with which the Neurological Society is concerned can often be approached both from the side of the nervous system and from the side of consciousness, and we must ignore neither the one nor the other of the two great highways leading to knowledge. It so happens that in the case of inhibition the first suggestion and first advance were made by studying the mental process. Herbart, seventy-five years ago, laid great weight on the arrest or inhibition of one idea by another, and developed mathematically an elaborate statics and dynamics of the mind. Obstructive association, it will be remembered, is a term occurring in English psychology, and recently much attention has been given to the subject by psychologists, both on the Continent and in England.

I have read Dr. Mercier's paper with great interest; but there is much in it with which I cannot agree. Analogies are in better place as illustrations than as arguments. I cannot admit Dr. Mercier's interpretation of "the first law of motion"; indeed it seems to me that he brings forward his own *reductio ad absurdum* when he says, in concluding, "(7) every nerve-centre controls simple centres, and is controlled by more complex centres than itself," thus picturing an endless series of nerve-centres always becoming more complex. It seems more reasonable to suppose that nerve-centres inhibit (using the word in its broadest sense) themselves and each other. It is indeed this fundamental property of the nerve-cell, that of storing up energy or inhibiting itself, on which all life and development depend. There could be no growth if all afferent impulses were immediately transferred into efferent; it is owing to the fact, that part of the energy coming from without is stored up in the body, that the individual grows and the race

develops. Looked at from the side of the mind, we see how in the lower animals, and even in savages and children, action is chiefly determined by the immediate surroundings, and we can note the gradual growth of control or inhibition, until in the developed man, thought, feeling, and action depend on the sum of impressions which have been inherited and gathered.

Our mental life is a continuous process of association and inhibition. We can only attend to ideas of a given degree of complexity, and we can only act in one out of a large number of possible ways. Thoughts, feelings, and actions are continually helping and continually hindering each other. The train of our ideas and the succession of our movements are the resultant of these forces. We cannot think of the shield as both silver and gold; we cannot both eat our cake and keep it. We may perhaps look on desire as motive, and will as the result of the conflict and inhibition of desires. Our character depends largely on the relation between motive and inhibition. Some yield readily to appetites and emotions; others have what we call self-control. Motives may be so carefully weighed against each other that no action follows, as in the case of Hamlet, who continually looked "before and after." Some drugs, as opium and alcohol, seem to paralyse the inhibiting power of the will. The refreshment from sleep is, doubtless, largely due to the rest of the inhibitory functions.

Inhibition is a subject so full of interest to the student of psychology, that I am tempted to say more; but in order not to prolong this discussion, I shall confine myself to calling special attention to the important papers on Inhibition, by Dr. Brunton in 'Nature,' and Dr. Gaskell in the 'Journal of Physiology.'

DR. MERCIER'S REPLY.

Underlying the whole of Dr. Jackson's remarks there is the assumption, that inhibition is exercised by all nerve-centres, and that the views I have advocated are correct. It would perhaps be more accurate to say, that this agreement indicates that I have correctly set forth Dr. Jackson's views. Granting that the results of the removal of inhibition vary with the rapidity with which the inhibition is removed, yet in any case the inhibition must first be there, and this is my contention.

I should scarcely regard the examples given by Dr. Warner as instances of inhibition. In the highly nourished and actively changing tissue of the young brain the general nervous tension is

normally high, and these vague undirected movements seem to me to be due to a bubbling over, as it were, of superfluous nervous energy. When a novel impression is made on the sense-organs, this energy is not so much inhibited as drafted off in new directions and utilised in other ways.

Dr. Broadbent's contribution to the debate appears to me extremely valuable. It is a great gratification to me to find that he agrees to the non-existence of special inhibitory centres, and of special trophic centres and nerves. His suggestion, that such structures as skin and muscle *are* the nerve terminals and not merely receive them, appears to me, from this point of view, absolutely correct. Considering the effect that nerve-lesions have upon nutrition, considering that the function of these organs depends on nervous transmission, and having regard to the fact that function and nutrition are two sides of the same process, it appears to me inconceivable that the constant wash of nerve-waves upon a tissue should not affect and regulate its nutrition.

With regard to his hypothesis, that the connection between the nerve-centres and the nerve-endings should be looked upon more as the disturbance of a tension than as the transmission of a force, I am unable to give to this so unqualified an adhesion. I would ask if there is not some danger of our being led astray by the difficulties of nomenclature, for after all what do we mean by a tension? Do we not mean a force that is struggling to escape? that is struggling to overcome a resistance? And is this not substantially the same state of things as I have suggested to exist in the nervous system?

In answer to Dr. Savage's question, I must confess that I am unable to draw any distinction between control and inhibition; and that the two terms, when used to connote a purely physical process, an affair of nerve-cells and molecules, may be used interchangeably. But control, so understood, has nothing whatever to do with moral control; and if it pleased any member of the Society to refer to this latter as moral inhibition, I know of no valid reason against his doing so. Dr. Savage must allow me to protest against his fathering upon me a theory, that the influence that travels in the nerves is a fluid. I speak of it always and everywhere as a force. If Dr. Savage was struck with a stick, surely he would be able to grasp the stick as a thing apart from the blow.

With regard to the hierarchical arrangement of the nerve-centres Dr. Savage warns me of the danger of pushing an illustration too far, and both he and Dr. Cattell consider, that the hierarchical hypothesis is altogether ruined by the want of any control for the highest centre of all. But the fact is that these difficulties present

themselves because the parallel was not carried far enough, and the analogy was not worked out to the end. If we pursue the matter to its conclusion, we shall find that the highest centre of all is represented not by the Commander-in-chief, but by the War Minister, or rather by the Cabinet, and that the control of this highest and most authoritative body is furnished, not by any single superior authority, but by the public opinion of the nation at large. It is the controlling check of the continual streams of information as the state of public opinion in the country, as to its resources, &c., and as to the relation to the country of affairs abroad, that determines the course of the Cabinet; and it is the controlling check of the streams of nervous energy arriving from the body at large, and from the outside world through the special sense-organs, that is the inhibiting agent on the highest of all nerve-centres. Even, however, if my explanation of inhibition should not be correct, I submit that the hypothesis of a series of nerve-centres, each controlling those below and being controlled by those above, is at any rate simpler and more comprehensible than Dr. Cattell's notion that nerve-centres inhibit themselves. How they can do this I must confess that I shall be unable to understand until I see Dr. Cattell get into a basket and lift that basket off the ground, or get into a wheelbarrow and wheel himself along.

Critical Digests.

ON THE MORBID ANATOMY OF FRIEDREICH'S DISEASE.

BY T. A. ORMEROD, M.D.

In the last few years, a considerable number of cases have been recorded, illustrating the clinical history of this disease, but very few observations upon its morbid anatomy. It may be well, therefore, to give a detailed abstract of three publications, in which new pathological observations are to be found.

We will begin with two cases by Rutimeyer.[1] These have been already noticed, from their clinical side in 'BRAIN';[2] having been the subject of a previous communication by Rutimeyer.[3]

CASE I.

Heinrich Kern, æt. 20.—Onset between 6 and 7 years of age, with simple ataxia first of lower, then of upper limbs. No affection of sensibility till the sixth or eighth year of the disease. Affection of speech, curvature of spine; finally, paralysis, and wasting of the legs, dorsal contraction of the great toes; diminution of cutaneous sensibility of the legs; nystagmus. Absence of patellar reflex; no affection of pupils, no pains or girdle sensations. Death from "general weakness," after the disease had lasted about fourteen years.

Nothing special to note in the post-mortem appearances; death was ascribed to fatty degeneration of the heart, and hypostatic congestion of the lungs.

Examination of the nervous centres after hardening in Müller's fluid shewed the brain and medulla to be uniformly wasted; the pia and arachnoid of the lumbar cord thickened and adherent; the nerves of the cauda equina and posterior nerve-roots thinned.

There was degeneration of Goll's columns throughout the cord; and in the cervico-dorsal region the inner parts of the cuneate fasciculi were involved, and in the lower dorsal region nearly the whole of them. The lateral columns were also degenerated, over areas corresponding to the crossed pyramidal and direct cerebellar tracts.

[1] Virchow's 'Archiv,' Bd. 110, p. 215.
[2] 'BRAIN,' No. XXV. Table I., No. 7. Table II., Nos. 18 and 20.
[3] Virchow's 'Archiv,' Bd. 91.

Measurements of the cord after hardening—
In mid-cervical region 14 × 8 centim.
„ dorsal „ 9 × 6 „
In lower dorsal „ 9 × 6 „
In mid-lumbar „ 10 × 9 „

MICROSCOPICAL EXAMINATION OF THE CORD.

Cervical regions.—Marked degeneration of Goll's columns, which was however, a little less intense near to (within one c.m. of) the posterior commissure.

Just exterior to Goll's columns was a tract of much less intense degeneration, broadest at the posterior periphery of the cord, and narrowing further forwards. In the upper cervical region this area of degeneration did not reach the posterior commissure; but lower in the cervical region it became broader and extended further forwards, so that only a narrow tract of white substance along the edge of the posterior horn was left unaffected.

In the lateral columns was an area of degeneration corresponding accurately to the cerebellar and crossed pyramidal tracts; the degeneration was of equal intensity through all this area; less marked than in Goll's column, but increasing in intensity from above downwards.

Grey substance practically normal; anterior nerve-roots normal; degeneration of posterior nerve-roots more marked in lower than in upper cervical region.

Dorsal region.—Almost the whole area of the posterior columns were degenerated, Goll's columns intensely; the part immediately adjacent to the posterior horns of grey matter least intensely. This widespread degeneration of the posterior columns did not begin to abate till the extreme lower end of the dorsal region was reached.

In the lateral columns, degeneration of the cerebellar and crossed pyramidal tracts was present.

In the grey matter degeneration of Clarke's columns (both cells and fibres), reaching its maximum in the lower dorsal region.

Degeneration of the posterior nerve-roots.

Lumbar region.—Diminution of the degeneration in the posterior columns; especially in their antero-lateral part (angle between commissure and posterior horns). The posterior root-zones, however, particularly in the upper lumbar region, were degenerated.

In the lower lumbar region there appeared in the columns of Goll, a small oval area, mesially situated, wherein the degeneration was less intense than at other parts of Goll's columns.

In the lateral columns degeneration of the crossed pyramidal tracts.

Grey matter mostly intact.

Still considerable degeneration of the posterior nerve-roots.

Sacral region.—Degeneration (fainter but appreciable) in the median and posterior parts of Goll's columns. Lateral columns intact.

The medulla was not examined.

Case II.

Bertha Kern (sister of Case I.).—Note of Sept. 1882. Age 14; onset of disease at age of 7. Ataxia of limbs and of trunk; slight nystagmus; speech not affected. Little if any affection of sensation, no pain. Slight dorsiflexion of great toe. In 1883 rapid appearance of a spinal curvature. Permanent dorsiflexion of both great toes. Although extremely ataxic, the patient could get about by holding on to things. In Sept. 1884 a feverish attack accompanied by great prostration and pains in the limbs, in which she died. Typhoid was suspected, but this was not a substantiated post-mortem. Fatty degeneration of the heart was found microscopically.

The dorsal cord appeared to be small. Measurements (after hardening) of

Mid-cervical region	12 × 8	centim.
Upper dorsal „	10 × 6	„
Mid-dorsal „	8 × 5	„
Lower dorsal „	7 × 6	„
Mid-lumbar „	9 × 6	„

The dura mater from the 7th dorsal vertebra downwards appeared infiltrated with blood, but there was no haemorrhage into the dural sac. Slight adhesions of dura and pia, and some diffuse thickening of the pia mater.

After hardening in Müller's fluid, there could be seen (naked eye) degeneration of the posterior columns, limited in the upper cervical region to part of Goll's columns, but lower down spreading out towards the posterior cornua; and in the posterior part of the lateral columns a wedge-shaped area of degeneration reaching from the mid-cervical to the lower dorsal region.

The medulla oblongata was small. The brain was normal.

Microscopic Examination.

Upper cervical region.—In the posterior columns marked degeneration of Goll's columns reaching forward to the posterior commissure. Some affection of the fasciculi cuneati, chiefly in their middle third. Degeneration in the lateral columns, less marked, corresponding to the joint area of the cerebellar and crossed pyramidal tracts, and uniform in degree over all this area.

Grey substance normal; some loss of fibres in the posterior nerve roots.

Middle and lower cervical regions.—Goll's columns most affected. In the fasciculi cuneati the degeneration spread outwards so as to leave intact only their anterior parts, and the parts immediately abutting on the posterior cornua and posterior nerve roots.

Grey substance normal; posterior nerve-roots highly atrophied.

Dorsal region.—Still further degeneration in the fasciculi cuneati; their anterior and exterior parts, which were free in the cervical region, being now encroached upon. Low in the dorsal region,

though the anterior parts of the fasciculi cuneati were involved, the stripe along the posterior grey horns, and a little triangular patch at the entry of the posterior nerve-roots were more healthy than the neighbouring parts.

In the lateral columns was degeneration of the crossed pyramidal and cerebellar tracts (as in the cervical region): but it is noted that in the upper dorsal region (and here only) an additional area was involved, reaching forwards from the anterior part of the cerebellar tract, along the periphery of the cord, to the level of the forward termination of the anterior cornua.[1]

Clarke's columns degenerated, especially at the lower part of the dorsal cord; the rest of the grey matter intact.

Posterior nerve-roots diseased.

Lumbar region.—Posterior columns; anterior parts of cuneati fasciculi normal again, while of their posterior parts a portion was affected, viz., the inner half of the area defined by Westphal as the zone of entry of the posterior roots; Goll's columns degenerated; the little oval zone of comparatively healthy substance which was mentioned in Case I., appeared in this case also. Lateral columns: less extensive degeneration, corresponding to the diminishing crossed pyramidal tract.

Degeneration of Clarke's columns in dorsi-lumbar region. Decrease in the degeneration of the posterior nerve-roots.

Sacral region.—Posterior columns: only the postero-median parts diseased. Lateral columns: degeneration of crossed pyramidal tracts still traceable.

The medulla oblongata could not be thoroughly examined; but degeneration could be traced in the funiculi graciles and cuneati to the upper part of the decussation.

A certain degree of degeneration was found in the nerve trunks (median and sciatic).

Histologically the spinal degeneration consisted (in both cases) of a growth of fine connective tissue replacing the nerve fibres; there was no special affection of vessels, and though the pia was thickened in places, there was no reason to think that the disease had spread inwards from the meninges.

The localisation of the disease moreover suggests that it was parenchymatous in origin: for it is obvious, as the author remarks, that it affected several distinct tracts of fibres, and was limited to these tracts: constituting therefore a "combined system disease." Thus in the lateral columns the crossed pyramidal tracts and direct cerebellar tracts were picked out with tolerable accuracy. (A slight extension beyond the areas of these tracts was noted in Case II.; for which we have suggested an explanation in the footnote.) This lateral degeneration was at its maximum in the lower dorsal region. The direct pyramidal tracts were not affected, Clarke's columns were diseased, which accords with the German view, that the cells of these columns are the trophic centres for the cerebellar tracts.

[1] This probably represents the antero-lateral ascending tract.—(Abstractor's Note.)

In the posterior columns, Goll's tracts were diseased throughout; excepting a small oval centrally placed area in the lower parts of the cord, which probably forms no part of Goll's tracts. Concerning the rest of the posterior columns it may be said that in the lumbar and lower dorsal regions the disease spread widely enough to involve the posterior root-zones, while in the upper cord it was limited to the neighbourhood of Goll's tracts. How far the changes in these parts, the fasciculi cuneati, are to be considered as "systematic" degenerations is doubtful, but they correspond broadly to those of ordinary tabes.[1]

Dr. Everett Smith[2] gives a clinical account of six cases of Friedreich's disease, and the morbid anatomy of one. The clinical histories should be read in the original; we can here only make the following brief excerpts.

Five sisters were affected with the disease: three other sisters of the family did not suffer from it: five brothers were also unaffected.

The symptoms in the girls are summarised by the author as "a series of morbid symptoms, beginning at the age of 6 or 7, regular in order in progression. The first symptoms noticed were gastric disturbances and palpitation of the heart, followed by an unsteady gait which later became purely ataxic. The ataxia gradually extends to the hands; and here the disease seems to remain stationary for an indefinite length of time."

We may add the following symptoms from the clinical histories of the girls: club foot, spinal curvature, *wrist drop roughly resembling that seen in lead-palsy*, attacks of epileptic convulsions; of pain in the spine and limbs; of dyspnœa and collapse. Oddly enough, the only girl whose symptoms did not begin with cardiac palpitation, became the subject, later on, of organic disease of the heart from rheumatism. In another girl the tendon reflex[3] persisted after the gait had become ataxic, though it disappeared subsequently.

The father of these girls suffered from an allied form of paralysis, but his case differed in the following respects.

(1) The symptoms began much later in life (æt. over 60). After an attack of rheumatism, brought on by exposure to cold, there followed a "severe gastritis." But he had had attacks of nausea and vomiting for many years. He had dilatation of the heart.

(2) Although the gait was ataxic, the tendon reflexes were present.[4]

(3) The symptoms disappeared under treatment.

This man's family was decidedly "neurotic"; his father (the girls' paternal grandfather) had some doubtful ataxic symptoms; and although the father of the five ataxic girls did not drink himself, there was a marked history of drunkenness in the two generations preceding him.

[1] For the minute localisation we must refer to the original paper.

[2] 'Boston Medical and Surgical Journal,' vol. cxiii. p. 361.

[3] Cf. a case shewn to the Medical Society.

[4] Cf. 'Brain,' No. 39–40. A woman with spastic paraplegia had two children suffering from typical Friedreich's disease.

The patient whose case was investigated post mortem died during one of the periods of collapse (above alluded to); it was accompanied by dyspnœa and palpitation, and vomiting so intense that she had to be fed per rectum. There had been in this case considerable paresis, in addition to the ataxy; and also affection of sensation in the lower limbs.

Post mortem, in the abdominal and thoracic organs nothing was found that bore upon the nervous disease. The muscles were generally wasted. The brain was not examined.

The spinal meninges were injected and adherent to the bony walls of the canal. The cord appeared "asymetrically atrophied, pale and in local areas softened."

The lower dorsal and lumbar regions were minutely examined, with the following results:—

The shape of the cord was altered, the posterior half being shrunken, and the left postero-lateral column looking as if it had been sliced away.

Posterior columns; their whole area intensely sclerosed, except a comparatively healthy part close to the posterior commissure. Rather more healthy nerve-fibres in lumbar than in dorsal regions.

Antero-lateral columns. Sclerosis of pyramidal tracts, crossed and direct, upon both sides, but of less intensity than that in the posterior columns.

Thickening of pia mater over the posterior and lateral columns.

Posterior nerve roots discussed; anterior healthy or nearly so.

Ganglion-cells of anterior and posterior cornua less numerous than normal, and showing degenerative changes under high powers.

Central canal blocked with round cells. In the lumbar region was a small supplementary canal just posterior to the main canal.

Dr. Pitt's case[1] is, we believe, the first reported in England in which the clinical history is supplemented by the morbid anatomy. The patient was under the care of the late Dr. Moxon on and off for seven years. He formed one of the family shewn to the Clinical Society by Dr. Gowers, and a notice of his case based on Dr. Gowers' paper appeared in a previous number of 'BRAIN.'[2] It should be added here that choreiform twitchings formed an early and a marked feature in his case, and that during the last years of his life failure in the organs of circulation supervened, as evidenced by dilatation of the heart, obstructive disease of some of the arteries of the limbs, bronchitis and dyspnœa. Death took place rather suddenly at the patient's own home.

Post-mortem.—The heart weighed 25 oz., both ventricles were dilated and hypertrophied, the left shewed fibroid patches, the aortic valves were diseased; the radial, on one side, could not be found, and on the other, contained an organised clot. The

[1] 'Guy's Hospital Reports,' vol. xliv., p. 369.
[2] No. XXV. (Table II. No. 31.)

author considers that there was obliterative end-arteritis of the small vessels.

In the brain, the only abnormal appearance was a tough and streaky grey patch "below the posterior part of the external capsule" in the left hemisphere.

Spinal cord small on section; measurements as follows:

Upper cervical region.	Normal cord.	Degenerated cord.
	13 × 10	13 × 8
33 mm. lower.	15 × 9	12 × 8
39 „	16 × 9	11 × 7
51 „	12 × 8	10 × 7
67 „	10 × 8	9·5 × 7
81 „	10 × 8	8 × 7
107 „	13 × 8	8 × 8
127 „	12 × 8	8 × 7

It was of firm consistence, and there was grey degeneration of both posterior and lateral columns.

The meninges were normal.

The nerve-roots—anterior, healthy—posterior, degenerated (confirmed microscopically).

Peripheral nerves—radial, and branch of anterior crural examined and found normal.

Microscopical examination of the cord.—Weigert's method, and aniline blue black employed).

Lower lumbar region.—Posterior columns completely sclerosed, except their anterior fourth, which is healthy, and a thin strip extending therefrom backward in the middle line. Internal radicular fibres sclerosed.

Crossed pyramidal tracts shewed loss of medullary sheaths.

Upper lumbar.—Posterior columns sclerosed, except a strip along each posterior grey horn (in the anterior half of the posterior columns).

The disease of the lateral columns reaches forward along the periphery beyond the area of the cerebellar tracts.

Lower dorsal.—Similar sclerosis of posterior columns. Also of Clarke's columns. Lateral columns much as before.

Mid-dorsal.—Posterior columns as before. More extensive sclerosis of lateral columns, involving the crossed pyramidal and cerebellar tracts; and (irregularly and slightly) the periphery in front of them. Clarke's columns affected.

Upper dorsal.—Goll's columns much sclerosed. Burdach's columns become more healthy, especially their anterior third near the grey matter.

The lateral sclerosis was most marked in the dorsal region.

In both dorsal and lumbar region there was partial disease of the nerve-fibres entering the posterior grey horn.

Lower cervical.—Goll's columns completely sclerosed; slight sclerosis of posterior two-thirds of Burdach's columns.

Sclerosis of crossed pyramidal tracts less; cerebellar tracts still affected; peripheral sclerosis well marked, extending up to and slightly along the anterior fissure.

First cervical.—No degeneration in the lateral columns.

In the medulla.—Sclerosis of funiculus gracilis; slight disease of funiculus gracilis, and of funiculus rotundus.

At the level of the decussation of the pyramids two canals were visible.

The author's summary may be given thus.

1. Spinal cord extremely small.

2. Extreme sclerosis of Goll's columns, through its whole length.

3. Burdach's columns severely sclerosed, their posterior portion particularly (the disease being rather less intense high in the cord). But the strip of Burdach's columns which bounds the posterior horn and root, especially along the anterior half, is free.

In connection with (3) slight disease of funiculi cuneati and rotundi in medulla?

4. Sclerosis of crossed pyramidal tracts, most marked in dorsal region (?), not traceable above decussation of pyramids. Much less intense than sclerosis of posterior columns.

5. Sclerosis of cerebellar tracts, also not intense.

6. Slight irregular peripheral sclerosis anterior to (5); sometimes reaching along the anterior fissure.

7. Degeneration (here and there) of Clarke's columns; of posterior nerve-roots, and fibres in the posterior horns.

The localisation of the degeneration in these cases throws some light on its nature: this must be our excuse for the above lengthy abstract. All previous cases agree in this, that the posterior columns have been deeply sclerosed; and generally in this way: that the columns of Goll (postero-median) have been affected through the whole cord, the columns of Burdach (postero-external; fasciculi cuneati) extensively affected in the lower dorsal region, and less extensively higher up the cord. Here therefore the degenerative changes roughly correspond to those of ordinary tabes, and if the latter be regarded as a "systematic sclerosis," so also (quoad the posterior columns) must Friedreich's disease.

But as regards the lateral sclerosis, there has hitherto been some doubt whether this follows definite nerve-tracts, developing in them independently of the posterior sclerosis, i.e., whether it is primary and systematic; or whether it is caused by irritation spreading from the diseased posterior columns, i.e., is secondary and diffuse. Now the present cases speak strongly in favour of the former view. For in all of them the degeneration picked out known tracts of fibres, the crossed pyramidal and cerebellar in the first two, the crossed and direct pyramidal in the third, the crossed pyramidal and cerebellar in the last. In the last case also an irregular peripheral sclerosis extended round the anterior parts of the cord in its upper parts. This is reasonably ascribed to disease of the antero-lateral tract (Gowers, Hadden, Tooth); an explanation which may account for the existence, in other cases of combined sclerosis, of a peripheral degeneration not corresponding to the cerebellar tracts. And since there is no evidence that the disease spread from the posterior to the lateral columns, either by con-

tiguity or in other ways,[1] the inference is that the process has been one of independent degeneration in several nerve-tracts; the antero-lateral sclerosis probably being secondary to the posterior in point of time, but in no other sense.

Further, the more localisation in tracts already marked out by development and function argues powerfully for the view, that the disease is ab initio parenchymatous and begins in the nerve-fibres. Dr. Pitt advances the view, that this nervous degeneration is itself dependent upon hereditary disposition to disease in the blood-vessels; for he finds an objection to what may be called the purely nervous view in the fact, that the most sclerosed areas (Goll's columns) are just those which develop later than the less sclerosed (crossed pyramidal tracts). But we hardly think it proven that the latest in development must necessarily degenerate first; for other factors may interfere with the working of such a law: e.g., as Dr. Pitt himself indicates, the posterior columns may break down first because they have more continuous work thrown on them. And, clinically, the vascular phenomena seem to have appeared late in Dr. Pitt's case; while some of those symptoms which he singles out in other cases as vascular, e.g., headache, vertigo, comatose attacks, may just as well have been nervous. But his remarks on the condition of the circulatory system are none the less valuable; for whatever their relation to the nervous disease, symptoms from this side must be of high prognostic importance.

A word may be added on the nomenclature of the disease. Dr. Everett Smith,[2] taking as text Dr. Gowers' remarks on the inconvenience of naming diseases after individuals, proposes for it the name "postero-lateral spinal sclerosis of generic origin." The term "generic" is ingeniously chosen to express a family grouping of cases, necessarily connoting transmission as "hereditary" seems to do. As a shorter name for ordinary use Dr. Smith proposes "generic ataxia." But here he would, we presume, be met by Dr. Pitt, who says that "the pathology and course of this disease and of ordinary locomotor ataxia are entirely distinct, the only common symptom being the loss of the knee-tap." We do not altogether subscribe to this latter statement, but at least it shews that till our views about a disease are thoroughly stereotyped it is difficult to find for it a rational name.

[1] It should be mentioned, however, that Dr. Everett Smith notes in his own case an inflammatory condition of the cells of the central canal like that observed by Brousse, and believed by him to represent the channel by which the disease spread from one part of the cord to the other. Dr. Smith's own opinion is that it spreads by means of meningitis. Seeing that the antero-lateral sclerosis in his case exactly corresponds to the crossed and direct pyramidal tracts, we hardly see how this can be maintained.

[2] 'Boston Medical and Surgical Journal,' March 1, 1888.

CHRONIC HEREDITARY CHOREA.

BY W. P. HERRINGHAM, M.D.

In 1842 Dr. Waters of Franklin, N. Y., wrote to Dunglison describing a peculiar form of chorea infesting certain families in that neighbourhood, which was remarkable in four points. It was markedly hereditary, it rarely appeared before adult life or after 45, it was incurable, and it in all cases induced dementia. Dunglison inserted the letter in his third edition, with the remark that he had never himself seen anything answering to this description.

The same disease was found by Dr. Charles Gorman of Luzerne, Penn.,[1] to prevail in other portions of the country also.

The next description is by Lyon,[2] who writes twenty years later that he has been familiar with it all his life, that it occurs in certain families only, that it is hereditary, and that it is called "migrims" by the people. He does not describe the disease in any detail, does not mention any mental affection as occurring with it, and in several instances states that it began in childhood. He gives three histories: (1) in which five generations, (2) and (3) in which three generations, were affected. Case No. 1 had two brothers who never had chorea, but one of them had two children "in whom well-defined chorea has existed for many years." He says, "To these cases many more might be added."

In 1872 Huntington[3] described a chorea hereditary in a few families in a part of Long Island. It affected males more than females, came on gradually, always after middle life, was incurable, and was usually accompanied by insanity and a tendency to suicide. If one of the family escaped, his descendants were ever after free from it.

Ewald [4] in 1884 described two cases in different families. There was the same strong hereditary character, but in neither case was the mind affected, nor does he mention insanity in any of the affected relations.

Clarence King[5] gives a family history of disease in four genera-

[1] 'Inaug. Dissertat. at Faculty of Jefferson Med. Coll. of Philad.,' quoted by Dunglison.

[2] 'Amer. Med. T.,' N. Y., 1863, vii. 289.

[3] 'Med. and Surg. Reporter,' Philad., April 13, 1872, quoted by Sinkler in Pepper's System, vol. v. 440.

[4] 'Zeitschrift f. klin. Med.' 1884, vii., Suppl. H. 51.

[5] 'N. Y. Med. Journ.,' xli. 468, 1885.

tions and a description. He lays stress upon the insanity which accompanies it.

Peretti[1] gives an elaborate family tree of his case; the whole family springs from Frau N., who was choreic. Two children were healthy, and had healthy descendants; two were diseased. They had between them fifteen children, of whom nine had the disease and four had other nervous affections. Of the nine choreics, one had four children over 30, none of whom were affected, and three of these had healthy children themselves; four others had children, among whom one male aged 23 and probably two boys were already diseased. Insanity is only mentioned in four of the nine.

Huber[2] had a case of a man aged 38: the disease began at 30 in his face, spread first to the arms, then to the legs. He notices that the movements, which otherwise resemble juvenile chorea, are easily controlled. The only mental symptom in this case was weakness of memory. The man's sister, aged 42, was choreic and demented; his father choreic and insane, his father's sister choreic. The disease was found occurring in four generations.

In the Collective Investigation Report[3] compiled by Dr. S. Mackenzie, West mentions a family in which three generations were diseased.

Zacher[4] describes a case and gives the family history, showing three and perhaps four generations affected.

Hoffmann[5] describes another set of relations similarly diseased.

Macleod[6] treated and dissected two sisters, who were choreic and insane; their father had died of a disease which made him shake all over, and two brothers are said to be choreic. It is to be hoped that more details will be forthcoming; for though the cases seem probably to belong to this category, they are exceptional in more than one point, and the family history is insufficient.

Lannois[7] traces the disease in four generations of one family, and describes six of the cases at length.

Ewald, Huber, Zacher, Hoffmann, and Lannois give most careful and excellent descriptions of the cases they observed.

From these authors a good picture of the disease can be constructed. It is a disorder of movement, affecting the voluntary muscles only, which exactly resembles chorea in appearance. Hoffmann does indeed describe a case (Jos. Kärcher) in which the movements were more like violent tetanic spasms; but it is so different in other respects also, that with all deference to Hoffmann I do not consider it right to class it with the present group. The disorder is hereditary, and affects more individuals of the families which it infests than most other hereditary diseases. Thus, taking only the patient's own generation, King has four affected

[1] 'Berl. klin. Woch.,' 1885, No. 52.
[2] 'Virch. Arch.,' cviii. 1887, 267.
[3] 'Brit. Med. Journ.,' 1887, i. 425.
[4] 'Neurolog. Centralblatt,' 1888, 34.
[5] 'Virch. Arch.,' 1888, 513.
[6] 'Journ. Mental Science,' xxvii. 1882, 191.
[7] 'Rev. de Méd.,' vii. 1888, 645.

out of five, Peretti nine out of sixteen, Huber two out of two, Hoffmann five out of nine (Wipfler family). In the second generation of Lannois' family five out of twelve were choreic. These five had fourteen children, of whom eleven were diseased. On the other hand, all agree that when once the chain is broken the disease does not recur. An exception to this is, however, to be found in Lyon's paper; but he does not seem to be speaking from personal knowledge. The character of the inheritance is therefore peculiar. The disease affects both sexes, and is heritable either through the female as in Ewald's, or through the male as in Huber's case. It nearly always begins at or after 25 years of age, but occasionally earlier. Lyon, an exception in this as in many other points, speaks of its beginning at puberty. Peretti has a man of 23 in whom it had already begun to show itself, and another who had twitchings from childhood, and died paralysed at 19; while Felic. Wipfler (Hoffmann) began to be diseased at the end of her school-days. Twitchings in the face are noticed in children of diseased families by Peretti and Zacher. Waters places the age of onset not before adult life or after 45; Huntington says it never comes on until middle life; Ewald's cases nearly all began at 30; King places the onset between 25 and 35; Macleod's cases began at 60 and 70.

The disease does not develop out of ordinary chorea; but begins without known cause by twitching in the face (King, Huber), spreading thence to the arms and later to the legs, or is general (Hoffmann) from the first. The movement is violent, and coarse in character, not like Friedreich's paramyoclonus, a fibrillar twitching insufficient to move the limb. In the legs it produces a curious gait: there is a sudden stoppage, the patient looks as if he was going to fall forward, sways his body violently, at last is able to take a few rapid steps, and so recovers his balance (King, Hoffmann, Lannois).

It is stated in several cases that coarse motor power is preserved. In Felic. Wipfler it was but small. It is not uncommon for ancestors to be described as having been "paralysed." In some cases the finer movements are impossible, but it is noticed by several that these patients are much more able than ordinary choreics to control the involuntary movements by the will, so that those who are in constant and violent movement can yet drink, eat, and dress themselves without difficulty. In most cases the movements cease in sleep, but Hoffmann noticed them even then.

There is no wasting of muscles, no anæsthesia; the deep reflexes are natural or somewhat increased, and the electrical reactions are normal.

There is no heart disease connected with the affection, nor is rheumatism an antecedent. The bodily functions are carried on naturally, and micturition and defæcation are, so long as the mind is sound, properly performed.

The disease is very commonly followed after a short time by some mental degeneration. This is noticed by every author except Lyon and Ewald. Though usual, it is not invariable. Two of Huber's cases were intelligent officials up to their death, and Ewald's cases were both perfectly sane. It not unfrequently

happens that other neuroses occur in members of the family who are not choreic (Peretti, Huber, Lannois). The choreic insanity begins with loss of memory and childishness, and goes on gradually to dementia. In two cases (Huber, Zacher) there were fits of violent destructiveness.

There have been three post-mortems recorded. Macleod (if his cases be admitted) found in one case a subdural hæmorrhagic cyst, with leptomeningitis over one side of the brain only, in another multiple tumours in the dura mater. Huber transcribes the post-mortem record of the father of his own patient. Pachy- and leptomeningitis were the only lesions. Another post-mortem, which will be of great importance, is to be expected from Klebs, who has dissected one of the cases which Huber reports in detail.

It is of the greatest interest to compare this disease with ordinary chorea. The movements are of similar character, and in both there is commonly mental affection. The one is closely connected with rheumatism and heart disease, and recovers. The other has no such connection and is progressive. The conclusion which the comparison suggests is that the two are produced by affections of the same parts, but of a different nature. On the other hand, it has certain analogies with general paralysis. Tremor and weakness in the one are replaced by loss of control and involuntary movement in the other; loss of muscular power supervenes at the end of some cases. The exalted delusions of the general paralytic are in some instances (Hoffmann) of this disease represented by cheerful misrepresentation of their real condition. The post-mortems show here, as in general paralysis, affections of the meninges.[1]

That it is an affection of the brain is a generally expressed opinion, and that it is impossible as yet to say more is obvious.

[1] De Witt, 'Cincin. Lanc. and Observ.,' 1876, xix. 408, has a paper on bilateral chorea occurring in a general paralytic. I have not seen it, but it sounds as if it formed a link.

W. P. Herringham.

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of recent Papers. By ALEX. HILL, M.D., Master of Downing College, Cambridge.

TEXT-BOOK.

1. Petit Atlas Photographique du Système Nerveux. J. LUYS (*one vol.*, 16*mo*, *Paris*, *T. B. Baillière*, 1888).

COMPARATIVE ANATOMY.

2. The Nerves and Vessels of Amnocoetes and Petromyzon Planera. A. DOHRN (*Mitth. aus der zool. Station zu Neapel*, viii. 2. pp. 233–307, *July*, 1888).

3. The Comparative Anatomy of the Brain. The Fore-Brain. EDINGER (*Abhandl. der Senckenbergischen Naturf. Gesellschaft*, 1888. *Separatabdruck*, *Frankfurt Diesterweg*; 4 coloured lithograph plates).

The disposition of the mantle, and the arrangement of those bundles of medullated fibres which are common to all vertebrate brains investigated by means of section series. Staining with Weigert's hæmatoxylin and carmine, or acid fuchsin.

In all transverse sections, two portions are to be considered; the thick ventral portion projecting up into the floor of the ventricle, and the thin roof and outer wall. The former is the corpus striatum or stem-ganglion, the latter the mantle. The mantle is always covered with a layer of grey matter, but a cortex in the ordinary sense is a late development in the animal series. Part of the mantle in cyclostomes and teleostei, is devoid of cortex, like the septum pellucidum of primates. The stem-ganglion is always nervous.

1. Amnocoetes—little round cells in small groups uniformly disposed through the whole thickness, separated by finely-fibrous substance which arranges itself into thicker fibre bundles, forming in front, a commissure and an olfactory chiasm, behind a commis-

sure (C. interlobularis), and a track running towards the mid-brain, but not ending there. The author regards it as a veritable basal fore-brain bundle. The fine plexus fibres take Weigert's hæmatoxylin almost as well as ordinary medullated nerves, although not surrounded by what we ordinarily regard as medullary sheaths.

2. Teleostei.—The mantle is so thin as to be usually overlooked. It presents no median fold similar to the longitudinal fissure of higher vertebrates. In front of the mid-brain it forms the sack-like recess for the pineal gland, owing to the great development of the mid-brain; the 'tween-brain is obscured, and has often been overlooked, but it has the usual ganglia and bundles of fibres. From the stem-ganglion a single medullated basal fore-brain bundle (pedunculus cerebri) originates. In developing Corvina it can be seen to originate by three roots, two from the more thickly grouped cells in the dorsal and lateral region, the third from a well-defined group near the median line.

In all vertebrates there is a commissure (or chiasm) between the basal fore-brain bundles in the 'tween-brain region. The commissura interlobularis (Ant. Com.) was usually non-medullated; some of its fibres unite the stem-ganglia, others come from the olfactory nerves. The crossed fibres are prolonged into the 'tween-brain. The stem-ganglion represents only the putamen and caudatus of higher animals, not the globus pallidus. It gives origin to fibres and is not intercalated in their course. In the same way the basal fore-brain bundle represents only that part of the pedunculus cerebri which springs from the corpus striatum.

3. Selachians.—The fore-brain varies very much in form, but usually presents a central fused mass with an unpaired ventricle, and two enormous lateral olfactory lobes. The homologies of the central mass are very obscure. To this problem the author devotes himself. By studying embryos it is found that the fore-brain consists almost entirely of anterior wall, this is greatly thickened and folded down anteriorly, constituting the primary unpaired fore-brain which alone exists in the rays; the ventricle disappears. From the primitive cerebral cavity, frontal-lobes bulge out in the sharks.

In adult cartilaginous fishes the fore-brain exhibits three groups of cells beside the nerve cells everywhere scattered through its substance. The dorsal stem-ganglion group gives origin to the basal fore-brain bundle; the ventral group to the anterior commissure, and perhaps to the olfactory nerve. In the "most occipital portion of the mantle" lies a third group which gives

origin to a "mantle bundle." The basal fore-brain bundle passes backwards, some fibres cross to opposite sides; some fibres end in the thalamus, others go on to the medulla oblongata. The mantle-bundle crosses to opposite side at base of fore-brain. This crossing has nothing to do with the often described commissura Halleri. The anterior commissure which connects the stem-ganglia contains but few medullated fasciculi in the rays; in Scyllium it is reinforced by considerable medullated tracts from the olfactory lobes. There is in addition a median bundle originating in front and dorsally, and passing downwards to the basal fore-brain bundle.

The amphibian brain is the simplest in the vertebrate series. By the backward prolongation of each cerebral hemisphere, occipital lobes are formed. The nerve fibres do not become myelinated until just before adult life; the basal longitudinal bundle passes backwards towards the medulla, some fibres, however, end dorsally in the 'tween-brain, and others in the regio infundibuli.

In reptiles an indubitable brain-cortex is met for the first time (there are probably in some part of the mantle both of amphibia and dipnoans, cortex formations). Author gives a description of this as found in different parts of the mantle and in different animals; it appears that the typical cortex formation only occurs in the occipital region, and at the dorsal angle of the median surface. Scattered round pyramidal cells are found on the outer wall of the cortex, in relation with fibres of origin of the olfactory nerve. A third kind of formation is found between the other two.

Cortex.

4. Comparison of the Convolutions of the Seals and Walrus with those of Carnivora, and of Apes and Man. Turner (*Journal Anat. and Phys.* xxii. pt. 4, pp. 554–582, *July* 1888).

Contains a repetition of work done for the Challenger Reports, including a detailed description of the brains of a large number of carnivora other than the Pinnipedia. Treats of the homologies of these brains among themselves, and with those of primates, especially the limits of the hippocampal lobe, and the Island of Reil which the author regards as situate on the general surface in carnivora, as the whole, or part of the Sylvian convolution. In the polar bear an entire arched convolution is concealed within the

fissure of Sylvius; the Sylvian convolution is partially concealed in the seals and walrus. This explains the apparently discrepant results which follow stimulation of the margin of the fissure of Sylvius in monkeys and carnivora. Author reviews the evidence as to the homology of the fissure of Rolando and crucial sulcus, and inclines to the belief that the former is represented by the coronal fissure of carnivora, and that the latter is of secondary value, and not necessarily represented in higher animals. The præsylvian fissure is the homologue of the præcentral; the intraparietal fissure is to be found in the anterior part of the lateral (second curved) of carnivora.

Localisation.

5. Communications on Localisation in the Great Brain, and their Practical Value. Jastrowitz (*Deutsche Med. Wochenschr. No.* 5, 1888).

Calls attention, more especially from the surgeon's point of view, to the number of difficulties and uncertainties which still stand in the way of diagnosis of brain lesions. Calls attention especially to two cases, in the first of which hemiamblyopia was associated with one lesion at the upper end of the ascending frontal convolution, and another at the junction of the gyrus fornicatus and præcuneus. In the second case no motor disturbance resulted from a tumor as large as a cherry in the ascending parietal convolution.

Cortex—Stimulation.

6. Landois' Experiments with Chemical Stimulation of the Brain. Lenbuscher and Ziehen (*Centralbl. f. Klin. Med. No.* 1, 1888).

Cortex—Excitability.

7. Co-existence of Inexcitability of the Excito-Motor Zone with Persistence of the Motor Functions of this Nervous Centre, and also with the Production of Violent Epileptiform Attacks. Brown-Séquard (*Comptes Rendus Soc. de Biol.* p. 354, *April* 14, 1888).

After considerable loss of blood the cortex becomes, as is well known, inexcitable. An animal in this condition, however, will move about normally without showing motor or sensory dis-

turbance. Stimulation of its cortex will sometimes throw it into convulsions.

Temperature of Cerebrum.

8. Temperature Changes in the Great Brain associated with Emotions. Tanzi (*Phys. Centralbl. No.* 3, pp. 57–64, *May* 12, 1888).

Thermo-electric investigations carried out in Schiff's laboratory at Florence.

Six dogs and two apes experimented upon; anæsthesia with morphia in the circulation and cocain applied locally. The thermo-electric needles sunk through trepan-holes until they touched or perforated the dura, but did not injure the brain. Various suggestive sensory stimuli were applied, and the place, time, and character of the alteration of temperature recorded. When the narcosis was too deep for changes in the brain, variations in the temperature of the spinal cord were observed, which sank to very small proportions when the brain variations began to set in. The thermal changes which resulted from "mind stimuli," whether on one or both sides of the body, affected the whole brain on both sides. They were not altered by faradisation of the sciatic nerve, or placing ammonia under both nostrils. The changes did not consist in a single elevation or depression of temperature, but in an irregularly rhythmic change. In the spinal cord the rhythm kept pace with the reflex movements; in extreme cases the variation reached 3° C. above or below the normal. The changes, judging from the effects of different stimuli, are proportional to the intensity of the emotion. They appear to be independent of change in circulation.

Corpus Callosum.

9. Cases of abnormally short Corpus Callosum Cerebri. Schroeter (*Allg. Zeitschr. f. Psychiatrie*, xliv. *Nos.* 4 and 5, p. 408, 1888).

Epiphysis.

10. Epiphysis Cerebri in Petromyzon fluviatilis. Whitwell (*Journal of Anat. and Phys.* xxii. pt. 4, p. 502, pl. xviii., *July* 1888).

Corrects in certain points the description published by Ahlborn.

Cerebellum.

11. Constituents of the Anterior Peduncles of the Cerebellum. Bechterew (*Archiv f. Anat. u. Phys.*, 1888, *Anat. Abth.* 2–4, pp. 195–205, pl. xiii., *June* 15).

Commences with historical account. Developmental method. Human embryos. Not fewer than four tracts can be distinguished by the periods at which their fibres become myelinated; 1*st.* (in fœtus 27–28 cm. long) has a shorter course than the others; 2*nd.* (at 33 cm.) is longer and lies in the dorsal portion of the half-moon shaped peduncle as seen in section carried between the cerebellum and corpora quadrigemina; 3*rd.* (35–38 cm.) smaller than the two former, lies in the middle of the half-moon; 4*th.* and longest (at end of intrauterine life) lies on the inner side of the others. The first is isolated from the others both at its origin in the cerebellum, and at the decussation; its fibres do not go into the cerebellum itself, but are lost in the neighbourhood of the nucleus nervi vestibularis (Bechterew) on the outer side of Deiter's nucleus. Forwards they only travel with the others as far as the front of the pons where they cross the middle line. The remaining three tracts connect the cerebellum with the great brain; the crossing seems to be complete. All three go into the red nucleus, but whether their fibres go round its cells, or are broken by them, undetermined. The second and third tracts are connected with the central nuclei and the cortex of the vermis, that is to say with the region connected also with the fibres of the posterior peduncle, except those coming from the under olive. Comparing their development with that of the tracts which make up the posterior peduncle, it is seen that the dorsal bundle (2) myelinates at the same time as the direct cerebellar tract; the middle at the same time as the fibres on the inner side of the corpus restiforme which originate in the nucleus nervi vestibularis, and the fibres from the nucleus of Goll's column.

Circulation.

12. The Brain Arteries and their Enclosure in Bony Canals. Rudinger (*Archiv für Anat. u. Physiologie*, 1888, *Anat. Abth.* 2–4, *July* 15, pp. 97–109, pt. vi.).

The diameter of the carotid arteries is not much more than one half that of the carotid canal. The relation of the vertebral arteries to the foramina in the transverse processes is about the

same. The rest of the space is occupied by the venous network. The pulsation in the arteries is a factor in propelling the blood in the veins; the arrangement of the veins cannot regulate the blood supply to the brain.

CIRCULATION.

13. Lymph-paths of the Brain. ROSSBACH and SEHRWALD (*Centralbl. f. d. Med. Wissensch.* 26 and 27 *June*, 1888).

The conclusions in this paper are based entirely upon the supposition, in which the authors considered themselves justified, that the method which Golgi trusts to for staining nerve-cells and their processes is really a method for filling up the perigang-lionic and perigliac spaces with silver precipitate. Interpreting the appearances which result when Golgi's method is used in this light, the authors give an account of the arrangement of the smaller lymph spaces and tracts in the brain. The figures, they say, which Golgi interprets as stained ganglion cells are always larger than the cells from the same piece of tissue as shown up by other staining methods. The form of the cells is different. No encircling space, such as is always to be seen with other methods, is visible around the dark figures. When Golgi's incrustation-mass is dissolved in ammonia the large figures give way to small cells of usual appearance. When the staining is incomplete pieces of axis cylinder, etc., are seen in perigangliouic spaces. The well-known uncertainty of Golgi's method depends upon the varying condition as to size and fulness with lymph of the lymph-tracts. The lymph spaces in the retina, stomach and elsewhere can be filled with precipitate of bichromate of silver by Golgi's method.

CIRCULATION.

14. The Influence of Simple and Epileptogenous Excitations of the Brain upon the Circulatory Apparatus. FRANÇOIS-FRANCK (*Comptes Rendus Acad. des Sciences, Paris*, cvii. 5. *July* 30, 1888).

CRANIAL NERVES—TASTE.

15. The Course of the Taste Nerve Fibres to the Brain, SALOMONSOHN (*Inaug. Diss., Berlin*, 1888).

Case of loss of taste over anterior two-thirds of tongue on one side, associated with loss of smell, blindness, and protrusion of eye-

ball on same side. Diagnosis of a syphilitic tumor extending from foramen opticum to foramen rotundum. Conclusion that taste fibres of lingualis passed via chorda, geniculate ganglion, great superficial petrosal, spheno-palatine ganglion to superior maxillary nerve.

Spinal Cord—Degeneration.

16. Lesions of the Spinal Cord produced by passing Anæmia of this Organ. Spronck (*Archives de Phys.* xx. pt. 1, p. 1, 1888).

Temporary ligature of the abdominal aorta induces permanent degenerations which only affect primarily the ganglion cells and their processes. The degenerations of the medullated fibres are secondary.

Spinal Cord—Nutrition.

17. Changes in the Spinal Cord which follow Occlusion of the Abdominal Aorta. Singer (*Sitz. der k. Acad. d. Wiss.* xcvi. pt. 3, p. 136, 1888).

The aorta was tied below the renal arteries in rabbits. Permanent sensory and motor paralysis usually resulted. The spinal cord was examined after varying intervals with the Weigert-Pal method. Almost all the cord degenerated with the exception of the posterior columns, roots and ganglia. The degeneration of the cells of the anterior brain was obvious.

Spinal Cord—Filum Terminale.

18. The Anatomy of the Spinal Meningies, the Sacral Nerves and the Filum Terminale in the Sacral Canal. Trolard (*Archives de Phys.* ii. 6, pp. 190–200, *August* 15, 1888).

The arachnoid cavity is not continued to the extremity of the sacral canal, but terminates at the second sacral vertebra. The first four posterior branches of the sacral nerves form a plexus giving rise to a posterior nerve. The last two in like manner constitute cutaneous and musculo-cutaneous branches, etc.

Reflex Action—Knee-jerk.

19. The Reinforcement and Inhibition of the Knee-jerk. Bowditch (*Boston Medical and Surgical Journal, May* 3, 1888).

Reflex Action—Respiration.

20. A Reflex Action on Respiration of Stimulation of the corneal branches of the Trigeminus. Guttmann (*Virchow's Archiv*, xciii. pt. 2, pp. 222–229, *August* 1888).

Boy of four years old poisoned with atropin—unconscious. Irritation of cornea with finger caused cessation of respiration in expiration lasting 5·9 seconds.

Senses.

21. A Kinesiæsthesiometer, with Remarks on the Muscular Sense. Hitzig (*Neurolog. Centralbl. Nos.* 9 and 10, 1888).

Method depends upon the measurement of the acuteness of the muscular sense by means of pieces of wood of the same size, but differently weighted with lead. The author finds that the ability to appreciate differences in weight sinks with the depression of general sensation in the limb, a conclusion which is opposed to the central apperception of the force discharged to do muscular work.

Peripheral Nerves.

22. Electric Organ of the Skate. Sanderson and Gotch (*Journal of Physiology*, vol. ix., Nos. 2 and 3, pp. 137–167, *August* 1888).

Histological account with figures. Authors do not believe in the nervous nature of the branched cells which are in relation with the terminal ramifications of the nerves. Minute and careful analysis of the electric phenomena of the shock, one hundredth of which is sufficient to throw the image off the scale of the electrometer, the maximum electromotive force being about half a volt. The current passes along the tail from before backwards.

Electric Organ.

23. Remarks on some New Researches on the Torpedo. E. du Bois-Reymond (*Sitz. der K. P. Akad. zu Berlin*, xxii. p. 531, 1888).

A criticism of the paper in Proc. Roy. Society (vol. xlii. p. 357, 1888) by Gotch.

Peripheral Nerves.

24. On the Distribution of the Cutaneous Nerves on the Dorsum of the Human Hand. Brooks (*Internat. Monatschr.* vol. v. pt. 8, 1888).

By means of dissections of the skin, with nerves attached, under water, the author shows that the nerves of the hand cross one another much more extensively than is usually supposed. Skin areas are supplied by more than one nerve. Hence partial retention of sensibility after section of particular nerves.

Psychology.

25. The Memory of Muscular Sensations. Beaunis (*Rev. Philosoph. No.* 5, p. 569, 1888).

Histogeny—Optic Nerve.

26. The Histogeny of the Retina and of the Optic Nerve. Falchi (*Archiv für Ophthalmologie*, xxxiv. 2, pp. 67–108, 3 plates, *July* 20, 1888).

A detailed account of the manner and date of formation of the several layers of the retina, etc.

Histogeny.

27. Development of the Substantia Gelatinosa Rolandi. Corning (*Archiv f. Mikrosk. Anat.* xxxi. pt. 4, *June* 14, 1888, 4 woodcuts).

The object of the investigation is to determine; (1) from which part of the original neural canal the substantia gelatinosa develops, or whether it is formed, as His believes, not from the neuro-epithelium at all, but from cells which have wandered into the neural tube from outside; (2) how soon it is distinguishable from surrounding parts; (3) whether the development throws any light upon the contrast which its structure presents to that of other parts. The author finds that it first appears as a local thickening of the inner layer of the grey matter in its dorsal portion. This thickening extends outwards on the dorsal surface towards the point of entrance of the posterior roots. It finally divides into one portion, which remains in the dorsal wall of the central canal, and the portions which permanently occupy the apices of the posterior horns. In new-born animals the substantia gelatinosa consists of

two kinds of cells. The first kind have large clear-staining nuclei with oval or spindle-shaped cell-bodies. The second kind have darkly staining nuclei and indistinguishable cell-bodies. This distinction into one class resembling nerve-cells, and another class resembling glia-cells, is still more apparent fifteen days after birth. The substantia gelatinosa comes from the cells which form the dorsal portion of the "inner plate." They do not lose their indifferent character until after both the anterior-horn cells and the grey matter of the posterior horn are differentiated.

Teratology.

28. The Brain and Eyeball of a Human Cyclopian Monster. Cunningham and Bennett (*Trans. Royal Irish Acad.* xxix., 1888, pl. 4 and 5).

Full term female child. No other deformities. Single lozenge-shaped opening, four eyelids. Mid and hind brain natural except no brachia to corpora quadrigemina, and no mesial geniculate bodies. 'Tween-brain distended, but epiphysis normal (although hollow); optic thalami fused; pulvinar absent. Single optic nerve arising from the base of the brain in front of the tuber cinereum. Fused cerebral hemispheres, the margin of the cortex above the mid-brain being raised into a narrow ridge ending on either side in a wider elevation corresponding to the fascia dentata. Corpus callosum, anterior commissure and fornix absent. Fused cerebral hemispheres terminated in front in a knob of cerebral matter about the size of a small bean protruding through frontal bone to bottom of left nasal pit. Authors look upon this as indubitably fused olfactory lobe. Optic nerve was single to its termination, but there were two retinal expansions forming two chambers within the eyeball. All the parts in front of the retina were double.

Method.

29. Carmine Staining for Nerve-tissues. Upson (*Neurolog. Centralbl.* ii. p. 319, *June* 1, 1888).

Three methods; (1) 5 c.c. Grenacher's carmine alum (1 gramme carmine boiled in 100 c.c. 5 per cent. solution of alum), 10–20 drops of acetic acid, 1–3 drops phospho-molybdic acid, stains nerve-cells, axis cylinders and connective tissue in 2–10 or more minutes: useful for over-hardened tissues; (2) carmine alum saturated with zinc-sulphate, same results; (3) water 4, alcohol 1, "carmine acid" 0·06, sections immersed 3–10 minutes. Colour fixed with

acetic acid (orange), saturated lead acetate (blue), sulphate of iron (black), manganese sulphate (red), nickel sulphate or barium chloride (violet). Axis cylinders, nerve cells, and connective tissue stained. Tissue must be well hardened.

Remarks on the foregoing, Krauss (*ibid.*).

METHOD.

30. Summary of the Osmic Acid and Gold Chloride Method. KOLOSSOW (*Zeitschrift f. Mickroskopie*, vol. v. pt. 1, pp. 50–53, 1888).

METHOD.

31. The Differences in Chemical Reactions of Nerve-cells. FLESCH (*Mittheilungen der Naturf. Gesellsch. in Bern*, 1888).

An attempt to bring together the well-known differences in the behaviour of nerve-cells to colouring reagents, metallic oxides (osmic and chromic acid), alkalies, acids, etc., with a view to classifying nerve-cells on a chemical basis. Cells grouped as chromophobic and chromophilous. Author establishes a relation between function and chemical behaviour; staining power resides in protoplasm, not in the granules. The chromophilous cells are as a rule smaller, contain dark granules, have a sufficiently alkaline reaction to yield Ehrlich's methyl blue reaction. When methyl blue is injected into the circulation of the living animal they stain like nerve fibres. They strongly reduce osmic acid. The protoplasm of the cells stains in carmine, etc., but not the nucleus (nor does the nucleus stain in chromophobic cells). Eosin stains the nuclei of all nerve-cells when they have been hardened in acetate of lead or other alkaline or neutral media. This distinguishes nerve-tissues from other tissues.

Pugibet on Paralyses in Dysentery and Chronic Diarrhœa of Hot Countries. (*Revue de Médecine*, February–April, 1888).—Out of eighty cases of dysentery and diarrhœa contracted in Tonquin, Dr. Pugibet relates *seven* in which nervous complications ensued. It is to be noted that all these patients came ashore in a state of great debility, insomuch that at least four of them died within a few days or weeks. The form taken by the "paralyses" was various. In two cases the muscles moving the head upon the trunk (excepting the sterno-mastoids) were affected, with both

deltoids and, to a less extent, the flexors and extensors of the forearms and wrists. In a third there was hemiplegia, and in a fourth, paralysis of the serratus magnus on one side, and the trapezius and the rhomboidei on the other. In the three other cases there were respectively, transient *glycosuria*, double trigeminal *anæsthesia*, with partial deafness; and finally paralysis of the bladder and rectum supervening, with a small patch of cutaneous anæsthesia, only twenty-four hours (!) before death. The special features of the motor paralyses were quick onset without cerebral symptoms, short duration and recovery beginning at the parts last (which were also least) affected. Several hypotheses as to the nature and seat of the lesions are discussed. The author cannot accept *ascending neuritis*. This theory, as explaining the paralysis following after diphtheria and other acute diseases, is not now widely accepted, Vulpian and others having declared the nerve lesions actually found to be secondary to a primitive lesion in the medulla. Dismissing peripheral conditions, whether of nerve or muscle, as inadequate to cause the symptoms under discussion, it is shown that they do not follow nervous distribution. Nor can they o n a reflex origin, like some rare paralyses due to uterine displacements or affections of the genito-urinary tract, for else they would observe some correspondence, in time or in severity, with the intestinal lesions. But this is not the case. M. Pugibet therefore attributes them to transient lesions of the anterior cornua of the cord; and cites two cases from Delioux in which, on autopsy, softening was discovered in the cervical and lumbar enlargements. The proneness of the post-dysenteric paralyses to affect isolated muscles, or muscular groups, or both upper or both lower limbs (according to various writers), is pointed to as a feature bringing these cases into the category of spinal affections; although it is admitted that they differ from ordinary cornual paralysis in non-febrile onset, short duration and absence of atrophy. The facial anæsthesia, deafness and glycosuria are referred to similar changes at the level of the fourth ventricle.

From the cachectic state of the patients, one might suppose that a simple failure of circulation in the nervous centres would account for the symptoms; but, on the other hand, in anæmia and chlorosis, although paralyses occur, they have not the special features seen in these cases,—sudden onset, often by night, rapid development, often a short duration and sometimes recovery coincident with aggravation of the cachexia. These diagnostic points distinguish the paralyses following a dysentery from those

commonly seen in convalescents, the "asthenic paralyses" of Gubler. In the Author's view the probable explanation of them is a thrombotic coagulation in the fine capillaries of the medulla, an event favoured by the cachectic condition of the blood and by the slowing of the circulation during sleep. The effects would vary with the degree of collateral circulation established, and according to the particular group of vessels primarily affected. Thus in severe cases local atrophy or softening may follow, leading to incurable paralysis. The previous history of marsh fevers in many of the cases naturally suggests a *miasmatic* cause. In these fevers nervous complications occur, either with the accession of the fever, or in the later cachectic period. Maillot, Jaccoud and others have already regarded paralyses in the first stage as due to an early congestion of the nervous centres, whilst Rindfleisch attributes them generally to melanæmia, causing obstruction by pigment, and rupture of capillaries among the nervous elements. Whatever be the pathology, the fact remains that paralyses of paludal origin are not rare, tend to appear and disappear with the attacks of fever, commonly take the form of right hemiplegia or aphasia, and that, like other symptoms due to the same cause, they readily yield to the influence of quinine. The post-dysenteric cases do not satisfy these conditions: but it seems probable that pigmental cells (present in all miasm-infected blood) may have contributed to the capillary lesions referred to.

In addition to his own seven cases already referred to, the Author cites ten more from other physicians: and shows how in these also the seat of motor affection is very variable; that sensory symptoms are not uncommon, especially after dysentery in the acute form; that electrical contractility is normal or slightly diminished, and that as a whole the symptoms presented are ordinarily incomplete and tend to disappear in the course of a few months. In a minority of cases they may persist, or, becoming generalised, may cause death by invading the respiratory centre. For treatment,—in the paludal cases Dr. Pugibet gives hypodermic injections of the hydrochlorate of quinine, of which he has administered as much as two or three grammes within a few hours.

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